ECEIVED
PR 16 1945
S. U. LIBRARY

MARCH 1945

UNITED STATES
DEPARTMENT OF THE INTERIOR
HAROLD L. ICKES, SECRETARY

BUREAU OF MINES
R. R. SAYERS, DIRECTOR

INFORMATION CIRCULAR

THE HAZARD OF HYDROGEN FLUORIDE POISONING IN THE MINERAL AND ALLIED INDUSTRIES

BY

R. R. SAYERS AND SARA J. DAVENPORT

.C. 7311,
arch 1945.

INFORMATION CIRCULAR

UNITED STATES DEPARTMENT OF THE INTERIOR - BUREAU OF MINES

THE HAZARD OF HYDROGEN FLUORIDE POISONING IN THE MINERAL AND ALLIED INDUSTRIES[1]

By R. R. Sayers[2] and Sara J. Davenport[3]

CONTENTS

Page
roduction 2
perties 2
story 4
osure to fluorine 7
  In the smelting industry 8
    Refining of aluminum 8
    Production of beryllium 11
    Magnesium founding 13
  In the petroleum industry 14
  In other industries 16
orine poisoning 17
  cute fluorine poisoning 17
  Chronic fluorine poisoning 18
xicity of hydrogen fluoride 21
  Permissible limits of hydrogen fluoride in air 23
thology of fluorine poisoning 24
ction of fluorine compounds in physiological processes 25
termination of chronic fluorine poisoning 28
  Examination of teeth for mottled enamel 29
  X-ray examination of the bones 30
  Analysis of the urine 30
termination of fluorine 31
evention of fluorine poisoning 31
  Plant operating precautions 31
  Personal protective devices 34
  Physical examination 36
eatment of fluorine poisoning 37
mmary 39
bliography 46

---

[1] The Bureau of Mines will welcome reprinting of this paper, provided the following footnote acknowledgment is used: "Reprinted from Bureau of Mines Information Circular 7311."

[2] Director, Bureau of Mines.

[3] Information specialist (Translator), Bureau of Mines.

Original from THE OHIO STATE UNIVERSITY

INTRODUCTION

The increasing use of hydrogen fluoride (annual consumption now excee 500,000 pounds (1)[4]) directly and its occurrence as a byproduct of the utilization of fluorine compounds in the mineral industries, especially in metallurgical processes and in the refining of petroleum products, has aroused interest in its toxicity, and a brief review of the literature, with special reference to the hazard of poisoning in the mineral industri seems to be opportune.

Recently, residents in the vicinity of aluminum plants have been complaining of injury to livestock and vegetation as well as to their own heal from the gases discharged into the air during operation of the plants, hydro fluoric acid gas apparently being the offender. The comparatively recent utilization of hydrofluoric acid in the alkylation process for the productio of high-octane gasoline has raised the question of the hazard of poisoning the petroleum industry. In addition, the use of fluorine-containing refrig ants has extended the potential exposure outside of industry (2).

PROPERTIES

The name fluorine is derived from the characteristic property of fluor minerals of acting as fluxes, that is, promoting the fusion of other mineral The fluoric substances of commercial importance are hydrogen fluoride and hydrofluosilicic acid; the minerals fluorspar (CaF_2) and cryolite (Na_3AlF_6); silicofluorides, which are byproducts of superphosphate manufacturing; and various synthetically made fluorides. The solid fluorine compounds usuall are colorless, crystalline, and amorphous substances (3).

As fluorine is one of the most active elements chemically, it does no occur free in nature. Fluorine compounds are found frequently in gases fr volcanoes and fumaroles. Igneous rocks usually contain this element in var ing amounts (0.01 to 3.36 percent), and its compounds have been found widel distributed in many types of rock formation. Small quantities occur in so of the micas, in topaz, and in tourmaline. Traces are present in sea water and some mineral springs; in bones, teeth, blood, and milk of animals, although it is said not to be a constituent of the human body at birth (4); and in plants (5). The principal fluorine-bearing minerals that are used in large-scale industrial processes are fluorite or fluorspar (CaF_2) cryolite (Na_3AlF_6), apatite ($3Ca_3(PO_4)_2 \cdot CaF_2$), and sedimentary phosphate rock. Fluorspar is used effectively as a flux in the smelting of many metals and in the ceramic industries (6). The largest users of fluorspar are the stee hydrofluoric acid, and glass and enamel industries, and it is employed extensively in insecticides, especially those for spraying fruits.

In the periodic system, fluorine is placed at the head of the halogen group. The element fluorine is the most electro-negative of all elements; its affinity for a number of other elements is very considerable. Free fluorine plays no part in toxicology, as it immediately reacts with water, oming hydrogen fluoride. Silicofluoride or fluosilicates are salts of ydrofluosilicic acid. Generally they are more readily soluble than the orresponding fluorides and in solution they give an acid reaction as hydrolysis occurs. Fluoraluminates - solids of the hypothetical acid H_3AlF_6 - re also of toxicologic interest; they are stable, sparingly soluble, and re not hydrolizable in aqueous solution.

---

[4] Figures in prarentheses refer to items in the bibliography.

According to Machle and his co-workers (2), hydrogen fluoride is a colorless gas (formula HF, molecular weight 20.01) which condenses to a colorless iquid at 19.5° C. It has a marked affinity for water, forms a fog with water apor in air, and when dissolved in water yields a constant boiling mixture ontaining 38.2 percent hydrogen fluoride which distills at 110.8° C. at 732 m. mercury and has a specific gravity 1.138 at 20° C. The molecular formula aries, in some instances being H_6F_6 (7); however, at partial pressures below 5 mm. mercury and at ordinary temperatures the formula may be taken as HF ith the molecular weight given above (2).

Hydrofluoric acid (hydrogen fluoride in aqueous solution) is a colorless, ming, mobile, corrosive liquid. It is prepared by treating fluorspar with oncentrated sulfuric acid in steel equipment at temperatures ranging from 00° to 400° F. (8).

According to Simon (7), the physical properties of hydrogen fluoride ive it considerable technical advantages over many other substances for imilar large-scale industrial use. Its low formula weight, 20, gives it ore effective chemical per pound than any other catalytic agent for use in he same reactions. This, of course, results in smaller and lighter equipment. As it freezes below solid carbon dioxide temperature, there can be ittle need for handling it as a solid. Its boiling point is conveniently ear room temperature, and it can thus be handled as either a liquid or a as without the need of using either high temperature or pressure. At 100° F. e vapor pressure of the liquid will give a gage pressure of only about 15 unds per square inch. The density of the liquid is nearly that of water t the same temperature, and it is thus more dense than most hydrocarbon aterial. It has a very low surface tension. This, with the high density, ables the liquid to mix readily with hydrocarbons by agitation but at the e time allows the mixture to separate readily and easily by settling. It ows little tendency to foam. Pipe and tubing of small diameter can be used, the viscosity of the liquid is low.

Two factors need be considered in relation to the use of liquid hydrogen uoride as a solvent. It has a high dielectric constant and is therefore a d solvent for salts. These salt solutions conduct the electric current. drocarbons and similar nonpolar liquids are not usually soluble in a liquid this type. Anhydrous hydrogen fluoride, however, is a very powerful acid, d basic substances are soluble in it through chemical interaction (7).

HISTORY

Fluorine minerals have been known since the Middle Ages. The Nuremberg artist Schwanhard (1670) is credited with the first attempts to etch glass b means of the vapors that are generated when fluorspar is treated with sulfuri acid. In 1771, Scheele produced the aqueous solution of hydrogen fluoride. In 1803, an Italian chemist, Dominico Morichini, demonstrated the presence of fluorine in a fossil elephant tooth found outside of Rome. In 1809, Gay-Lussac and Thenard described the corrosive effect of the acid upon the skin and showed that fluorine is present also in fresh teeth of animals and man. About 1846, George Wilson, the Scottish chemist, determined that fluorine is widespread in nature, in springs and sea water, in vegetable ash, and in blood and milk of animals (3).

It is only within comparatively recent years that the serious nature of fluorine toxicity has been realized, particularly as to chronic effects (9). It is from this viewpoint that the industrial use of fluorine is important. The possibility of a health hazard from fluorine compounds should be recognized wherever they are employed in industry or discharged into the air as waste gas.

Fluorine poisoning of human beings, animals, and vegetation by waste gases discharged from smelters, where the element is present in the ore or is used as a flux in roasting metals, has been a possible, although often unrecognized, hazard for many years. At one time the very existence of the smelter industry in Great Britain and Germany is said (10) to have been threatened by the many burdensome laws and regulations enacted to force the adoption of measures to prevent discharge into the atmosphere of harmful gases. In 1907, Ost (10) stated that an animal disease, consisting of diarrhea, anemia, and atrophy of the bones, which existed in the neighborhood for 20 years, brought the greatest danger to the Freiberg (Germany) smelte In 1855, the Freiberg smelters had to pay the first damages to the injured inhabitants. These damages reached the maximum of 61,000 marks in 1864, an in 1870, 18,000 marks were paid. Later, the damages amounted to 3,000 to 4,000 marks a year, until to 1893 a total of 880,000 marks had been paid fo current injuries and 644,000 for permanent relief (10).

Many of the cases of poisoning mentioned in the literature have result from oral ingestion of fluorine compounds, although poisoning from inhaling fluoric dust and hydrofluoric vapor has occurred. Roholm (3) mentions acut pulmonary symptoms in workers after they had inhaled fluoric dust. German workers in an electrolytic plant manufacturing beryllium exposed to dust of sodium silicofluoride and sodium berylliumfluoride as well as to active gaseous fluorine compounds (HF, SiF_4) developed symptoms of fluoric poisoning (Frostad (3) described fluorine intoxication among the workers at a long-established Norwegian aluminum works. Investigators handling fluoric compounds have suffered from protracted diseases of the air passages, and the Belgian chemist Louget died when he was 32 years old of a pulmonary affection probably due to exposure to such compounds (3). Roholm (3) found in th literature mention of only two cases of acute poisoning from inhaling gaseo fluorine compounds. Both were workers in a superphosphate factory in Dubli and were reported by Cameron (11) in a publication in 1887.

During the years 1911-1918, a disease like osteomalacia (softening of
e bones) appeared in the vicinity of aluminum works in Switzerland, the
iology of which was not clear. After long litigation, the works had to
mpensate for smoke damages to vegetation but were declared not liable for
e cattle disease, as expert witnesses considered the affection to be an
inary osteomalacia (3). However, Cristiani, who had been called in as
expert when the disease had almost disappeared, and Gautier (3) expressed
e hypothesis that the affection was chronic fluroine poisoning produced by
abnormally high fluorine content of the vegetation close to the works, as
result of the emanation of gaseous fluorine compounds (HF, H_2SiF_4). The
sease occurred within a distance of 4 to 5 kilometers of the works.
istiani and his collaborators (3) were able experimentally to produce
ronic fatal poisoning in guinea pigs by means of ingested fluorine com-
unds and to obtain the same picture by feeding fodder reaped near the
ctory on the windward side and fodder that had been exposed experimentally
hydrofluoric vapor. The cattle disease sometimes ended in death in the
urse of months. About one-third of 450 cattle were attacked.

In 1912, Bartolucci (3) described a strange disease among cattle on a
rm close to a superphosphate factory in Italy. The cattle became thin and
e coat coarse and lifeless; the animals lay down and got up with difficulty,
mped, and stood with curved, stiff backs and stiff hindquarters. Tender
ellings appeared at the joints and, after 4 or 5 months, there was thicken-
g of the ribs and bones of the head and shoulders. Finally, a cachetic
ndition (general ill health and malnutrition) developed, and the cattle
d to be slaughtered. Bartolucci (3) described the disease as osteitis,
nforming clinically to classical osteomalacia, and associated it etio-
gically with the content of fluorine compounds in the waste gases from
e works.

Probably the most spectacular instance of the ill effects of fumes dis-
arged from factories was the fog disaster in the Meuse Valley in Belgium.
the beginning of December 1930, a thick mist lay over large parts of Bel-
um, and several thousand cases of acute pulmonary attacks, with 60 deaths,
curred during the period December 3 to 5 in the densely populated valley
st of Liege. A Belgian investigating commission concluded that the deaths
re caused by poisonous products in the waste gases. Although it was im-
ssible to indicate any definite substance, the commission was of the opinion
at the offender was sulfur dioxide or its oxidation products, quantities of
ich were found in the factory smoke.

In 1931, Storm van Leeuwen of Leiden (12) advanced the theory that the
use of the Meuse Valley disaster was gaseous fluorine compounds emanating
om certain smelters in the region that were treating zinc ores containing
uorine. Fenner (13), who had followed the matter with great interest,
sumed that HF, and especially SiF_4, could play a part, and the publication
van Leeuwen's article strengthened his opinion, as the symptoms observed
re those of hydrofluoric acid poisoning. According to Fenner (13), the
uorine escaped for the most part as SiF_4 and not as HF. SiF_4 is especially
composable in water, but not quantitatively, and undecomposed silicon

5

fluoride can be present in the atmosphere. He mentioned, also, that H_2SiF_6 and the still less known fluorhydrine might have been present. Fenner remarked that if hydrofluoric acid has a toxic as well as an irritating action in this instance it appeared to be a question of compounds whose acid action was considerably slower than their toxic.

In a discussion in 1937 of the cause of the Meuse Valley disaster, Roho (14) refers to the lack of knowledge of the toxicity of fluorine compounds a that time and reviews some of the important literature that has appeared sin 1930 in confirmation of the opinion that the Belgian disaster was due to flu rine compounds. In this review, Roholm (14) stated that the presence of gas ous fluorine in the area affected was beyond question. Fifteen of the 27 factories in the area handled raw materials containing fluorine or added flu rine compounds to the raw materials and, consequently, were capable of discharging gaseous fluorine compounds from their smoke stacks. The 15 industr mentioned comprised 4 very large iron works with blast furnaces and steel wo 3 large metal works, 4 glass works and ceramic factories, 3 zinc works, and superphosphate factory.

In the winter of 1931-1932, during an examination by Flemming Moller a Gudjonssen (15) of workers in Copenhagen for silicosis, the X-ray revealed a pecular sclerotic bone affection in cryolite workers. In pronounced cases, the affection had attacked all the bones of the organism, but particularly the vertebral column and pelvis. The investigators assumed the affection t be fluorine poisoning caused by cryolite dust. The gastric symptoms were considered due to a slight corrosion of the mucous membrane of the stomach from the liberation by the hydrochloric acid of the stomach of hydrofluori acid from the dust swallowed. The bone changes were considered due to abso tion and the resulting deposition of calcium fluoride in the bones. Experi ments on rats revealed that cryolite is toxic, but symptoms identifiable wi either the acute or chronic poisoning were not observed.

In 1934, Slagsvold (16) reported the presence of disease in cattle gra around Norwegian aluminum works. The symptoms were loss of appetite, emaci ation, and anemia. Conjunctival irritation occurred with photophobia. Th gait was stiff and painful and bone fractures were common. The cows yield little milk. Sheep had thin, stiff wool that fell off considerably in win In both animals, especially in sheep, chewing was impaired. The grinding faces of the molars wore irregularly and formed sharp points correspondin hollows in the opposite teeth. In air-dried hay from the district as much 25 mg. fluorine in 100 g. was found. It was possible to show experimental that the affection was due to fluorine ingestion (17).

In 1935, Bardelli and Menzani (18) reported that since 1929 damage to vegetation, failure in the cultivation of silkworms, and disease among her vora of the character described above had been observed around an aluminum factory in northern Italy. Air-dried hay from the neighborhood contained average of 59.3 mg. of fluorine in 100 g., compared with 14 mg. in contro hay.

A peculiar condition of the teeth in different parts of the world, re-
rred to in the United States as mottled enamel, has been mentioned at vari-
s times in the earlier literature, but the trouble was not definitely con-
ted with exposure to fluorine until in 1916 Black and McKay (19) reported
e results of their study of the disease in Colorado. A similar dental dis-
se called darmous, which attacks the permanent teeth, of animals especially
t also of human beings, occurs in North Africa (Algeria, Tunisia, Morocco).
e disease is endemic in certain States of the United States, in South America,
na, Holland, Spain, Italy, Mexico, South Africa, Bahama, and the Cape Verde
ands (20). The first description of its occurrence was published by Velu
1) in 1922, although it was known earlier. Velu showed that it was not a
al dental disease but a general chronic intoxication by fluorine as a re-
lt of drinking water that had passed through beds of phosphorite having a
h fluorine content. The disease did not occur in places where the fluorine
ntent of the soil was lower than 20 mg. in 100 g.

In 1928, Kaminski (22) reported the complaints of workers in Petersdorf
stly those engaged in etching glass) of the roughness of their teeth due
exposure to hydrogen fluoride gas fuming in air which irritated the mucous
mbranes and injured the enamel of the teeth. He was asked frequently by the
rkers to whiten their teeth. He stated that the cause of the discoloration
s the death of the pulp and the consequent loss by the dentine of its trans-
rency due to effusion of blood into the pulp cavity, discoloration of the
eth appearing almost immediately.

According to Greenwood (23), recent biological interest in fluorine com-
nds is due essentially to the following observations: (1) The consumption
a drinking water containing 1 part per million, or more, of fluorine during
e calcification of the teeth causes a hypoplasia of the teeth (mottled
mel) in man and lower animals; (2) the feeding of certain phosphatic mineral
pplements containing considerable quantities of fluorine compounds to domes-
c animals has affected them adversely; (3) small quantities of fluorides re-
rd fundamental enzymatic processes; (4) in the United States, the use of
orine compounds (cryolite and barium fluosilicate) as insecticidal sprays
r fruits and vegetables has been expanded.

EXPOSURE TO FLUORINE

In addition to the uses of fluorine in large-scale industrial processes,
ch as in the smelting of many metals and in the production of glass, enamel,
d brick, their use on a small scale in the technical processes listed in
ble 1, prepared by Greenwood (23), may be possible sources of poisoning.

In an investigation by the Adult Hygiene Division of the Ohio Department
Health, assisted by the personnel of the Work Projects Administration in
io (24), contact with fluorine and its compounds was indicated in the
cupations given in table 2.

A more detailed review of the literature dealing with the hazard of
uorine poisoning in a few of the smelting and petroleum industries follows.

TABLE 1. - Uses of fluorine compounds in technical processes

1.	Binder for emery wheels	CaF_2
2.	Bleaching of cane for chair seats	HF
3.	Bleacher in laundries	Na_2SiF_6
4.	Cleansing graphite	HF
5.	Coagulating rubber	Na_2SiF_6; $MgSiF_6$
6.	Decolorizing of glasses	Na_2SiF_6
7.	Disinfection of hides and skins	H_2SiF_6
8.	Disinfection of hose and tanks in breweries	H_2SiF_6; $NH_4F.HF$
9.	Electrolytic manufacturing of beryllium	Na_2SiF_6
10.	Fixing of tannin on cotton in dye works and printing works	SbF_3
11.	Flotation of fluorspar and quartz	CaF_2
12.	Flotation of lead-zinc-fluorspar ores	CaF_2
13.	Flux	Na_3AlF_6; Na_2SiF_6; CaF_2
14.	Glass etching	HF; NH_4F
15.	Hardening cement	$MgSiF_6$; $ZnSiF_6$
16.	Insecticidal powders	NaF
17.	Making glue, paste and adhesives	HBF_4
18.	Making synthetic stones	Sundry compounds
19.	Optical industry	CaF_2
20.	Preserving wood (building) timber, telegraph poles	NaF; ZnF_2; $MgSiF_6$
21.	Recovery from phosphate plant	H_2SiF_6; $PbSiF_6$
22.	Refining mineral oils	SO_2F_2; Sf_6
23.	Refrigerating industry	CCl_2F_2; $C_2Cl_2F_4$
24.	Removing sand from castings	HF
25.	Rust removal from steel and iron	HF
26.	Silicate analysis and condensing agent in laboratory	HF
27.	Silk dyeing	HF
28.	Synthetic production of cryolite	CaF_2
29.	Treating anthracite for making gas coal	HF
30.	Window cleaner	H_2SiF_6
31.	Wood staining	CrF_3

Fluorine Exposure in the Smelting Industry

Refining of Aluminum

As has already been indicated in the section on "History," exposure to fluorides may accompany the reduction of aluminum inside the plant as well as through pollution of the surrounding atmosphere. The process employed for the production of metallic aluminum is virtually the same as that described in the original Hall-Heroult (25) patents of 1886-1888. The alumi made today is a result of the electrolysis of alumina (Al_2O_3) dissolved in bath of molten cryolite. Carbon electrodes dip into the fused salt bath container in a carbon lined steel tank or "cell."

TABLE 2. - Occupations in Ohio where contact with fluorine and its compounds is indicated

toclave men (chemicals).
aders (tin and enameled ware).
owers (glass factory).
ewmasters (distilled malts and wines).
rners (tin and enameled ware).
r men (fertilizer factories).
emists (distilled malts and wines).
n men (fertilizer factories).
ppers (tin and enameled ware).
evator men (fertilizer factories).
amel makers (tin and enameled ware).
chers (other manufacturing plants; electrical machinery).
aporator men (chemicals).
rmenting cellar workers (distilled malts and wines).
reman (distilled malts and wines; jewelry).
osters (electric fixtures).
rniture cleaners (dry cleaning and dyeing).
t galvanizers (electrical machinery).
secticide mixers (soap factories).
ttle men (distilled malts and wines).
Laborers (electric fixtures; tin and enameled ware; foundries; glass factories).
Machinist (storage batteries).
Mill men (distilled malts and wines).
Mixers (tin and enameled ware; fertilizer factories; glass factories).
Pit cutters (fertilizer factories).
Platers (electrical machinery; metal furniture; printing; brass factories; storage batteries).
Pressers (glass factories).
Processors (electrical machinery).
Rimmers (tin and enameled ware).
Rug cleaners (dry cleaning and dyeing).
Smelters (tin and enameled ware).
Spongers (tin and enameled ware).
Spotters (dry cleaning and dyeing).
Sprayers (tin and enameled ware).
Storage men (distilled malts and wines).
Tinners (electro-plating).
Washhouse men (distilled malts and wines).
Washing-machine operators (laundries).
Weigh-car operators (tin and enameled ware).

Aluminum oxide has a heat formation of 378,000 calories and therefore is e of the most difficult of the common metallic oxides to decompose (25). vy (25) first attempted the reduction in 1807, using metallic sodium or tassium. Theoretically, alumina can be reduced by carbon but, as a temperare of $2,100^{\circ}$ C. must be attained for the reaction to go on at a reasonable eed, major difficulties would be encountered (the metal boils at $1,800^{\circ}$ C.). 1854, Bunsen found that the fused chloride could be decomposed, but it reined for C. M. Hall, in 1886, to invent the present process whereby alumina dissolved in an igneous bath, composed mainly of aluminum fluoride, and en electrolyzed (26).

A standard electrolytic cell consists of a steel shell or pot about 8 et long, 5 feet wide, and 2 feet high, constructed of 1-inch steel plate, d lined with baked carbon 6 to 10 inches thick (this constitutes the cathode the system). Cylindrical carbon anodes are suspended from the anode bus r. Each pot consumes 8,000 to 20,000 amperes at a potential of 6 volts. dinarily there are 30 to 100 of these pots in series, making a total potntial drop of 200 to 600 volts. The igneous bath, maintained at about

900° C., is composed of approximately 59 percent of aluminum fluoride, 21 pe cent of sodium fluoride, and 20 percent of calcium fluoride. It has a speci gravity of about 2.1 and will dissolve up to 20 percent of aluminum oxide, a though it is usually maintained at 2 to 5 percent. As electrolysis proceeds molten aluminum collects in the bottom of the container in a layer 1/2 to inches thick, from which it is tapped as needed. An 8,000-ampere cell will make about 40 pounds of metal per hour; a 30,000-ampere cell, about 150 poun Theoretically about 5 kilowatt-hours are required to produce a pound of alum num, but actually 12.5 to 13 kilowatt-hours are necessary.

Under ordinary conditions, the Hall (26) process seldom produces metal containing more than 99.7 percent of aluminum. Impurities, largely silicon, copper, manganese, and tellurium, come mainly from the electrolyte, the alu and the carbon anodes. A cell to refine aluminum electrolytically, using a fused salt electrolyte, was patented by William Hoopes in 1901. This cell i still used. The cell itself and the lining and electrodes differ only sligh in design from the conventional aluminum pot. The distinguishing feature is the igneous bath, which consists of three distinct layers; on the bottom is aluminum anode of impure metal, next the igneous salt bath, and finally, flo ing on top and in contact with the cathodes, the molten cathodic layer of re tively pure aluminum. The anode material is a copper-aluminum alloy, low in titanium and iron, and containing some silicon to lower the freezing point. The igneous bath is made up of cryolite, aluminum fluoride, barium fluoride, and aluminum oxide to give the requisite specific gravity and fluidity. The bath is operated at a temperature of 900° to 1,100° C. The average metal pr duced will contain 99.8 percent of aluminum, but it is possible to produce a metal as pure as 99.98 percent, the small amount of impurities being copper, iron, and silicon.

To summarize: The principle of the method is the decomposition, under the influence of the heat developed by the electric current, of the cryolite and its combination with the silica of the carbon of the electric arc to fo a gas - silicon fluoride - which in turn is transformed almost immediately fluosilicic and hydrofluoric acid. The hydrofluoric acid thus produced may affect not only the plant personnel but persons within a radius of several miles (27).

Although the bath is not decomposed by an electric current unless the voltage is excessively high, there is a certain constant loss of fluorine d to volatilization and to the formation of incrustations on the lining. It i reported that the liberation of fluorine in aluminum reduction works is rev by the slight etching of the glass windows by the fluorine gas (27); this s indication of the presence of fluorine in the atmosphere may be noticed som times in the vicinity of aluminum plants.

The principal exposure of fluorine poisoning in aluminum plants is in working around the pots - adding the charge, changing electrode connections. renewals, and similar operations that require opening the doors of the pots for a short period at regular intervals. When the doors are opened wide, th hot currents generated by the hot bath carry most of the fumes out into the room where the workers are exposed unless each pot is properly ventilated, u til the fumes are carried away by ventilating currents and discharged throu roof monitors.

In plants with mechanical ventilating systems, large quantities of fumes are discharged from the roof monitors and from the stacks. Most of these fumes descend to the ground level and are deposited on the vegetation within a certain radius of the plant, in accordance with the prevailing direction of the air currents, and constitute a hazard to animals eating the vegetation.

Production of Beryllium

The economic importance of beryllium has led recently to its increased production and use. Opportunities for exposure to beryllium fumes and dust are fairly numerous in the industry in virtually all stages of beryllium production and in the manufacture of beryllium oxide and its subsequent use in the copper-beryllium alloys. There has not been, however, a great deal of experimental investigation of the health hazards probably owing to the comparatively small number of workers engaged and to the conflicting nature of the experimental data on the toxicity of beryllium available in the literature.

In 1936, Gelman (28) stated that the few experiments that had been made on the action of beryllium and its compounds were intended principally to determine the chronic action of beryllium introduced over a considerable period in small oral doses in the form of beryllium carbonate or chloride. He mentioned only two investigations in which an attempt was made to reproduce industrial conditions.

In the first of these, reported by Weber and Engelhardt (29) in 1933, it was noted that the nature of the illnesses of the workers exposed to various fluorides, among them beryllium oxyfluoride, produced during the foundry process of beryllium extraction, recalled to them the picture of poisoning by corrosive gases (chlorine, phosgene). This picture led them to conclude that the important toxic element was gaseous hydrogen fluoride. They proved experimentally that the dust of sodium siliceous fluoride and siliceous flux is highly irritating to the respiratory tract and has a toxic effect. Barium fluoride and beryllium oxide were much less irritating, inhalation of their dust being followed by virtually no observable disturbance to the health of the animals or any pathological-anatomical changes in the lungs. The authors (29) concluded that the question of the origin of the illnesses of workers in the foundry had not been clarified, largely because the vapors of beryllium oxyfluoride could not be used in their experiments.

The other investigation, carried out on dogs and rabbits at the Obuch Institute (30), Moscow, in 1934, revealed that 5 BeF_2 and pure metallic beryllium (both in the form of dust) caused no pulmonary lesions. Beryllium oxyfluoride, however, when smelted produced vapors that provoked serious pulmonary affections, but the characteristic action of these vapors could not be attributed to the action of the fluorides alone or to that of the beryllium oxide alone.

From his own analysis of the clinical phenomena and conditions present during the foundry method of extracting beryllium from its fluorides, Gelman

(28) concluded that the toxic effect was due to the vapors of beryllium ox fluroide and that their special physical condition (great dispersion) explained their characteristic toxic effect upon workers at a distance (even the open) as well as upon those close to the source of the vapors.

In 1943, the United States Public Health Service reported (31) the results of an investigation "to measure and define the toxicity of berylliu and to indicate, as far as feasible by means of animal experiments, the ma mum permissible concentration of dust or fumes from the metal or its compounds which workmen should be permitted to breathe." Results of the expe ments with ingestion by mouth, inhalation of dust and fumes, and peritonea injection of beryllium and its compounds indicate that beryllium of itself is not toxic. As no particular toxicity was established for beryllium, it appears from these experiments that whatever poisoning has occurred from e posure to beryllium salts has been due to the toxicity of the acid radica such as the fluoride or oxyfluoride, or to an objectionable condition bro about by the hydrolysis of certain of its salts, such as the chloride and sulfate. The investigators concluded that safe permissible working standa should not be based upon beryllium itself. Safe operating conditions in t preparation of the metal or its alloys must be based upon other considera than an implied toxicity of beryllium.

Examination of the gases and fumes evolved during electrolysis of beryllium revealed that the fluoride content varied between 0.07 and 5.0 mg. and the beryllium content varied between 0.03 and 0.73 mg. per cubic foot. Sodium, beryllium, and barium fluorides were used. The fumes given off co sisted of a true vapor phase and a very fine smoke or dust. Neither metal beryllium nor beryllium oxyfluoride was found in the dust.

With a single exception, all guinea pigs were killed within 90 minute or less when exposed to the dust and fumes. The toxic effect apparently w due to the fluoride dust and fumes, as the elimination of any one metal fr the melt did not lessen the toxicity appreciably.

The Bureau of Industrial Hygiene of the Pennsylvania Department of Health in 1944 reported (32) the results of an investigation of the health hazards incident to the manufacture of beryllium oxide in Pennsylvania and its use in the production of copper-beryllium alloys, which are employed i the manufacture of precision instruments, springs, and nonsparking tools.

A cursory examination of the medical records of the reported illnesse among plant workers in a month (September) selected at random indicated th 36 lost-time cases reported to the plant physician for treatment. Twenty-three of these (64 percent) were treated for dermatitis or a chest involve ment and the remaining 13 (36 percent) were treated for injuries incurre through accidents. Of the illnesses treated, 14 were for dermatitis and 9 for chest involvement. Absenteeism was high - about 6 percent - and turn averaged close to 100 percent a year.

The fact that employees exposed to fluorides in the plant still were reporting with respiratory diseases and dermatitis and that these disease appeared more frequently than others indicated that fluorides probably were the cause. As fluorine plays an important role in the extraction of beryllium oxide from its ore, and the oxide retains a certain amount of soluble fluoride, the fluorine is present in many of the departments of the plant.

Thirty-seven atmospheric fluoride samples were collected and analyzed for fluorine, which, calculated as sodium fluoride, averaged 0.996 milligram per cubic meter of air.

Magnesium Founding

The literature on the hazard of fluorine poisoning in magnesium founding is meager. The following data are taken from reports of investigations in two magnesium foundries, one made in 1942 and the other in 1944.

Under pressure of the demands of the war industries, production of magnesium has been greatly stimulated, and large-scale use of magnesium alloys has resulted in an increase in the health hazards peculiar to this industry. Among these hazards is the use of any of a large group of soluble fluoride salts as inhibitors and of ammonium acid fluoride, ammonium borofluoride, or ammonium silicofluoride as fluxes in melting (33).

According to Williams (33), the chief exposures to fluorides in magnesium foundries are the shakeout, molding, melting, and core spraying. Sulfur, boric acid, and fluorides are used as oxidation inhibitors to treat sand in making sand molds, in proportions ranging from 4 to 10 percent by weight, depending on physical factors in molding and casting. In core molding, core sands may be treated by mixing small quantities of sulfur and boric acid with the sand, or the finished cores may be sprayed with an aqueous (about 20 percent) solution of a fluoride, or both.

During the melting of magnesium by the crucible method (33), which is most commonly used, the metal is protected from oxidation by a fluoride flux. Fluoride is sprinkled on top during melting and is stirred in after all the metal is molten (about 65° C.). The charge is then heated to about 900° C. and removed from the furnace to a station where the dross is skimmed and a mixture of sulfur and borax (or some other inhibitor) is added. During this operation there is a brief but violent evolution of gases and fumes. The metal then is allowed to cool to pouring temperature of 700° to 800° C. Individual furnaces usually are ventilated to control fluoride fumes.

The presence of inhibitors in the sand is responsible for considerable air contamination during the shakeout operation. Fluorides in the sand are released, and high concentrations may be found in the air in the vicinity of the operation. This is usually the greatest single source of fluorides in a magnesium foundry, especially where there is a centralized shakeout. As the castings are broken out a mixture of steam, fluoride fume, magnesium oxide fume, dust, and various gases is given off. The amount of fluorides released during this operation depends on the size of the castings, concentration of the shakeout, amount of inhibitor in the sand, and the temperature

435

of the castings when they are broken out. The concentrations of fluorides found by Williams at the various points of exposure are given in table 3.

TABLE 3. - Concentrations of fluorides in a magnesium foundry

	Shakeout	Molding	Melting	Core spraying
Number of samples	14	32	10	3
Range of concentrations found (mg. per 10 cu. m. expressed as fluoride)	9.4-180	4.4-106	7.4-17.6	5.8-9.4
Average	87.7	18.8	12.6	7.0
Average deviation	±47.1	±14.5	±3.5	±2.0

In a study of the relationship between the urinary excretion and the bodily retention of fluorine, observations were made by Largent and Fernea (1) in three magnesium foundries. In view of the report of Williams (33) that the greatest atmospheric contamination was in the shakeout area, Lar and Ferneau expected to find the highest urinary fluoride concentrations samples provided by men working in that area. However, they found the hi est concentration in samples from men in the core-spraying and pouring ar although these areas have extensive ventilation. The core spraying is d in spray booths and, in addition, the men are provided with respirators. the atmosphere did not seem to be irritating, the workmen may not have w their respirators consistently; the elevated fluoride concentrations in t urine possibly may have indicated laxness on their part.

Largent and Ferneau (1) concluded from their investigation that flu exposure is occurring in some foundry areas but not in others, and that some instances fluorine undoubtedly is being retained in the tissues but a relatively low rate.

Fluorine Exposure in the Petroleum Industry

The paraffin alkylation reaction (8), by which isoparaffin and olefi hydrocarbons interact to produce highly branched paraffinic motor fuels, discovered by Ipatieff and Pines (34) more than a decade ago at the Rese and Development Laboratories of the Universal Oil Products Co. (34). Si the discovery of this fundamental reaction, extensive laboratory and pil plant experimentation has been carried on to study the reaction with sev catalysts. Investigations by Grosse and Linn (35) indicated that concentrated hydrogen fluoride was an excellent catalyst for alkylating isobut with propylene, butylene, or amylenes. The olefins were completely conv in the presence of an excess of isoparaffin to give a saturated product, excellent antiknock properties, and boiling almost entirely in the aviat gasoline range.

According to Gerhold and his co-workers (8), although the catalytic properties of hydrogen fluoride were attractive, the commercialization o

Digitized by Google Original from THE OHIO STATE UNIVERSITY

the process was delayed, largely because of the nature of hydrogen fluoride and the lack of experience in handling it in the manner and quantities required for hydrocarbon processing. When it became apparent that the facilities for the production of aviation gasoline would have to be expanded to supply the war needs, the development of the hydrogen fluoride process was rapidly accelerated. Pilot-plant tests for the investigation of charging stocks, operating conditions, and materials of construction, and process and mechanical designs of commercial plants that normally would have required years, were forced through in months.

Two major advantages of hydrogen fluoride alkylation over the older sulfuric acid process are the higher process temperature and the ease of catalyst regeneration. The higher temperature permits use of cooling water to remove the heat of reaction and thus eliminates critical refrigerating equipment. Catalyst regeneration is an inherent feature of the process; catalyst consumption is so low that the raw material and plant requirements for catalyst manufacture are considerably smaller than those for a sulfuric acid alkylation unit on the basis of equal alkylate production. Elimination of the problem of spent catalyst disposal, which is serious in many refineries using sulfuric acid alkylation, is a further advantage gained by fluoride catalyst regeneration.

Recently, a patent (No. 2,356,357) was granted to Schlesman and McCracken (36) for treating raw gasoline with boron trifluoride or hydrogen fluoride as catalysts. The raw gasoline, relatively high in olefin content, is treated with the catalysts mentioned at 300° to 500° F. and under 1,000 to 1,500 p.s.i. pressure, according to the claims. The treatment, it is asserted, reduces greatly the acid heat of the gasoline, a condition of primary importance, especially for aviation fuels. This means essentially that the olefin content of the fuel is reduced greatly, either by saturation or by polymerization to heavier products, which may be eliminated from the final product by distillation. Little has been published about the use of boron fluoride as a catalyst, although it has been studied extensively in research laboratories and is said to show some unusual results. Boron trifluoride, BF_3, is a gas at ordinary temperatures, boiling at -101° C., (-149.8° F.), whereas hydrogen fluoride, HF, boils at 67° F., or about room temperature.

The first commercial hydrogen fluoride plant was put in operation in December 1942, and several more were built during 1943 (37). When used as a catalyst, hydrogen fluoride may be a poisoning hazard unless it is confined within the processing equipment; therefore, new measures had to be adopted and rigidly enforced to protect the personnel of plants employing hydrogen fluoride (38). The principal sources of poisoning are from accidental spills of acid in the plant area due to failure to evacuate properly, to failure of the seals on pumps, contactors, or valves, or to rupture of lines, or vessels. There is also the possibility of accidental discharge of acid which may injure those exposed thereto. Burns may result from handling tools that may have been in contact with the acid. There is a possibility of pump and packing failures when adjustments are made to operating conditions. Repairmen may be exposed to high concentrations of hydrogen fluoride while repairing an extremely large leak.

Benson (38) states that it is almost impossible for anyone consciously to inhale enough hydrogen fluoride to harm himself seriously; the acid gas is too pungent for voluntary inhalation. The possibility of being rendered unconscious and becoming injured appears to exist only if a man is trapped when gases are released suddenly.

Fluorine Exposure in Other Industries

There are other industries and occupations in which a possible fluorin poisoning hazard exists, such as welding, pickling metals, manufacturing hydrofluoric acid and fertilizers, and many other less important occupation from the standpoint of number of persons exposed.

In a study of welding fumes with stainless-steel electrodes, Drinker and Nelson (39) found that the fumes contained 17.6 percent fluorine and that, theoretically, hydrogen fluoride could be evolved from the coating of the rods. However, they burned a great many such electrodes in response to complaints about the irritating nature of the fumes without finding hydrogen fluoride, although fluoride compounds were present. According to Ready (40), fumes from these rods may carry silicon fluoride, which, when inhaled, breaks down into hydrofluoric acid and silicon dioxide, and continued breathing of even slight concentrations of fumes from these special arc welding rods may cause fluoride poisoning.

Pickling baths containing 2 to 5 percent hydrofluoric acid in solutio or barium fluoride in suspension are used extensively in the sheet-metal industry for removing coatings of scale. This process is particularly adapted to cleaning aluminum or dural parts of airplanes before they are painted (40). Fluorine vapors from the vats may be a source of poisoning if measures are not taken to prevent their inhalation by employees.

In 1938, Wolff and Kerr (41) reported a case of chronic fluorine pois ing in a worker who had been exposed almost daily for 18 years to finely ground rock phosphate dust containing 3.88 percent fluorine.

It is interesting to note that a cement company (42) recently has adapted a process for manufacturing superphosphate to defluorinating raw rock phosphate, which is materially helpful in meeting a critical shortage in the bone meal necessary to the expanded livestock-raising program. The present supply of animal bone, most of which used to be imported from Sout America, falls far short of the demand, and much experimenting has been done to process rock phosphate for use as a substitute. Raw rock phospha cannot be used for this purpose as it contains too much fluorine, which is toxic to animals when present in large enough quantity. The Florida pebbl phosphate used contains 3.76 percent fluorine, whereas the defluorinated product is guaranteed to contain a maximum of 0.3 percent and actually con tains 0.08 to 0.12 percent (42). The pebble phosphate is pulverized and mixed with sulfuric acid. The reaction produces heat, steam, and dust. The operation is enclosed as tightly as possible, and an exhuast fan carries the dust and fumes to a wood stack, where water sprays bring them down in solution or suspension. The liquid contains about half of the fluorine and probably some sulfuric acid. It would appear that unless care is taken to protect completely the employees from the dust and fumes, a fluorine-poisoning hazard may exist during the defluorinating process.

According to the Oil, Paint, and Drug Reporter (43), six different plants are producing on a permanent basis defluorinated phosphate rock by processes developed in cooperation with the Agricultural Research Administration. These processes have been used by manufacturers in preparing nearly 100,000 tons of feed phosphate virtually free of fluorine.

Numerous cases of accidental poisoning by ingestion of insecticides containing fluorine are recorded in the literature, but apparently no information is available regarding poisoning of persons eating fruit and vegetables that have been sprayed by such insecticides. However, it has been stated recently (44) that the Food and Drug Administration proposes to limit the fluorine content of insecticide residue on apples and pears to 7 mg. of fluorine, calculated as F, per kilogram of apples or pears. The grounds for this proposed regulation are that fluorine and many of its compounds are poisonous and deleterious substances and, therefore, the quantity of insecticidal residue remaining on the fruit should be as small as practicable.

FLUORINE POISONING

Fluorine poisoning may be acute or chronic. Data on the subject reveal (20) that the form of fluorine used, the method and length of time of administration, as well as individual susceptibility, vary the toxic effects so much that comparison of the data is scarcely warranted.

Acute Fluorine Poisoning

Acute irritation of the gastrointestinal tract, such as vomiting (often with blood), diffuse abdominal pains, and diarrhea, appear very soon after ingestion of toxic compounds. Symptoms due to absorption occur after varying, usually short, periods as alternating convulsions or spasms and paresis, which may be localized or general. The spasms are very painful with pains and paresthesia in the extremities. Between the spasms general weakness and sluggishness are present. Various other less constant symptoms given by Roholm (3) are rise in temperature, difficulties in speech and swallowing, incoordinated movements of the eyeballs, thirst, salivation, and perspiration. In most cases death occurs after a few hours under prostration, dyspnea and failing pulse. Consciousness is unaffected.

When the intoxication is not fatal, there are signs of toxic nephritis (albumin, blood, and casts in the urine).

A single subcutaneous injection of sodium fluoride in the rat produces disturbance in the formation of the teeth, which, according to Roholm (3), appears macroscopically on the incisors after about 4 weeks as a circular, distinctly delimited band of white, unpigmented enamel. Subacute poisoning is manifested in the rat by the development of a cachetic condition with spontaneous hemmorrhage from the mucous membranes.

The effects of gaseous fluorine compounds is known only from rather limited material (1), local irritation of the mucous membranes predominating. Dyspnea seems to be the principal symptom in man. The acute toxic effects experienced by employees exposed to fluorides in a beryllium plant (32) were respiratory diseases and dermatitis.

The following characteristic symptoms have been listed by McClure (20) A condition of drowsiness and weakness resulting from paralysis of the vaso motor centers; cramps (epileptic in character), which may attack a single organ or the entire body; paralysis of the vasomotor centers; acceleration and deepening of the breathing, with paralysis following; vomiting; secretion of the salivary and tear glands, which is not stopped with atropin; early rigor following death. According to McNally (45), rabbits dusted daily with a mixture of 1/2 ounce sodium fluoride and 5 pounds silica dust suffered loss of weight, stiffness of the vertebral column, and finally paralysis of the spinal cord.

Sollman states that (46) acute poisoning presents chiefly the phenomen of acute gastroenteritis and collapse and may run a rapid course, with sudd nausea and vomiting, abdominal burning, diarrhea, for 3 to 6 hours, with stupor and weakness for 36 hours, in sublethal cases. Fatal cases develop muscular weakness and tremors, progressing to clonic convulsions originatin in the cord and higher centers, fall of blood pressure, general collapse, sometimes with grayish-blue cyanosis, dyspnea, and arrest of respiration and of the heart. Death may occur in a few minutes or may be delayed for 10 hours or longer (46).

According to Williams (33), with exposure to concentrations of fluori fume in excess of 100 mg. per 10 cm., a severe "biting" sensation is exper enced in the nose, followed within a few minutes by a "running nose" with discharge of considerable fluid. After about 20 minutes, blood may show o blowing the nose. This may persist for several days and in some persons m recur almost indefinitely. He states that these effects are rarely import enough to report to the plant hospital.

Chronic Fluorine Poisoning

In spite of the voluminous literature on the effects of exposure to fluorine on vegetation and animals and the demonstration of chronic fluorine poisoning in a dog by Brandl and Tappeiner as early as 1889, chronic fluorine poisoning in man is said not to have been known until it was discovered in 1931 by Moller (47) while he was investigating silicosis among workers in a cryolite factory. Radiological examination of the men for silicosis revealed peculiar changes in the bones of some. It was this dis covery that led to the researches by Roholm (3) and the publication of his comprehensive work on fluorine intoxication.

The symptoms of poisoning in the men examined by Moller (47) corre-onded clinically and pathologically with those found in animal experiments nd the radiologic findings obtained during life tallied with the post-mortem aminations made by Roholm (47) on two cryolite workers.

Attention was first called to "an endemic developmental imperfection of he teeth heretofore unknown in the literature of dentistry" by Black and cKay (19) in 1916. This condition they termed "mottled teeth." McKay (48) as the first definitely to associate the etiology of this condition with mething in the drinking water. In 1925, he (49) persuaded the community f Oakley, Idaho, where the incidence of mottled enamel was 100 percent, to btain its water supply from a nearby area where mottling had not been found. nother survey after 8 years revealed no mottling in the permanent teeth of he children born after the change had been effected. Chemical analysis of he water showed that the drinking water from the first source contained 6 .p.m. of F and that from the second source 0.5 p.p.m. of F.

It was not until 1931, however, that fluorine was demonstrated defi-itely by three independent studies (48) as the etiological factor in the ottling of the enamel. Churchill (50) found 2 to 13.7 p.p.m. fluorine in ater in endemic areas where mottling was observed. Smith, Lantz, and mith (51) confirmed Churchill's finding and demonstrated it experimentally n white rats. In the same year, Balozet and Velu (52) made similar obser-ations in the rock phosphate areas of North Africa.

Changes in the teeth are among the most easily reproducible and recog-izable of all the symptoms of chronic fluorine poisoning (3). These changes ave been studied by many investigators, whose results are in general agree-ent. Fluorine has a detrimental effect upon the growth of the teeth and the arts of a tooth calcifying during the period of fluorine intake will acquire ermanent defects; however, teeth or parts of teeth that are already calcified o not change with the administration of fluorine, except in high concen-rations.

Roholm (3) observed the following effects upon the teeth of rats ex-osed to fluorine. Rats are particularly valuable for this purpose as their ncisors grow continuously from a persistent pulp. With the lowest concen-ration of fluorine that causes an effect, the incisors of the rat lose the igment that normally causes their dark-orange color. The enamel becomes ighter, loses its luster, and finally becomes chalky white. Higher con-entrations cause localized hypoplasia (defective formation) of the enamel, hich becomes less resistent and chips off. The strength of the teeth is educed markedly, and the sharp, chisel-like cutting edge of the incisors ears off. Sometimes the teeth are worn off down to the gums. All incisors re elongated, especially in the upper jaw, where they curve back and may enetrate the roof of the mouth.

The lowest degrees of fluorine effect (3) produce alternate rings or ands of pale pathological enamel and dark normal enamel. Often the rings re arched, with the concavity towards the point of the tooth. The same henomenon is observable (3) when sodium fluoride is added to the drinking

Original from THE OHIO STATE UNIVERSITY

water. Intermittent injections of sodium fluoride cause the development alternating bands of pathological and normal enamel - a light and a dark layer for each injection.

The most interesting and probably the most significant symptoms of fl rine poisoning, from the health standpoint, are the changes in the osseou system. After absorption, fluorine is stored in the body, especially in t bones and teeth. This fact was first established by Brandl and Tappeiner (53) in an experiment on a dog. The condition was first noted in man by Moller (47) in 1931. As mentioned previously, Moller observed a peculiar appearance of the bones of men he was examining radiologically for silicos Roholm (3) found several bone changes in all cryolite workers examined wh had been exposed to much dust. He summarized the result of his radiologic examination as follows:

> The first thing to emphasize is the fact that the affection is a system-disease, for it attacks all bones, though it has a predilection for certain places. The pathological process may be characterized as a diffuse osteosclerosis, in which the pathological formation of bone starts both in periosteum and in endosteum. Compacta densifies and thickens; the spongiosa trabecula thicken and fuse together. The medullary cavity decreases in diameter. There is a considerable new formation of bone from periosteum and ligaments that normally do not calcify or only in advanced age undergo a considerable degree of calcification. All signs of bone destruction are absent from the picture.

In more than half of the workers (thirty-five) examined clinically b Roholm (3), motility in columna (ability to move the spinal column spontaneously) was restricted, both flexion-extension and rotation. The work were divided into three groups according to the degree of restriction. I the slightest cases there was merely a little stiffness of the lumbar and thoracic parts with slight restriction of extension and rotation; 21 pers belonged to this group. The lumbar and dorsal but not the cervical regio of the moderate cases was rather markedly rigid. It was possible for the as a rule with some difficulty, to pick up an object from the floor by be ing the knees; there were 10 workers in this group. Among the most sever cases (four elderly men), there was virtually complete rigidity of the en tire spine and more or less pronounced restriction of head movements. Th was particularly marked in a 57-year old male worker, who had been employ at the factory for 12 years. The head was fixed in a bent-over position; movement was possible only to an extent of 5° to 10° in both sagital and frontal planes. To look sideways, he had to turn the whole body. This m was unable to pick up anything from the floor by bending the knees and c not put on his socks without aid.

On the whole, the restriction of movement was found by Roholm (3) t parallel the osseous changes. It was noteworthy, however, that only som of the workers whose motility in columna was moderately restricted compl of stiffness. Even those most severly affected complained of surprising little inconvenience therefrom.

The most common chronic clinical symptoms of which the employees examined by Roholm (3) complained were various dyspeptic phenomena, shortness of breath, and rheumatic attacks. The gastric symptoms (loss of appetite, nausea, vomiting, and cardialgia) were experienced by 80.9 percent of the workers. The first three symptoms were definitely connected with the work and were experienced for a few days or weeks after beginning work in the dusty atmosphere. Thereafter some of the workers tolerated the dust, whereas others still had transitory symptoms after holidays or if the dust content of the air increased temporarily. Seven of the workers complained of continuous constipation or constipation alternating with diarrhea, which was more or less definitely connected with working in dust. Twenty-two workers complained of chronic gastrointestinal symptoms.

Fifty-one percent of the workers complained of circulatory and respiratory symptoms. Dyspnea was the most frequent symptom, usually moderate in degree on exercise but increasing in severity during exposure to thick dust.

More than one third of the workers complained of indefinite rheumatic pains and stiffness in the body that, with more or less justification, could be connected with affections in bones, joints, or muscles.

The most frequent nervous symptoms were tiredness and indisposition, which were experienced by 15 employees - 12 men and 3 women.

TOXICITY OF HYDROGEN FLUORIDE

Machle and his co-workers (2) found from their animal experiments on the inhalation of high concentrations of hydrogen fluoride that exposure to atmospheres containing 1.0 to 1.5 mg. hydrogen fluoride per liter produced death n a significant proportion of animals exposed for 5 minutes or more. Concentrations at or above this level may be considered dangerous for exposure f any duration. Although no deaths occurred in two groups exposed to 0.79 and 0.50 mg. per liter, respectively, for 5 minutes, 7 of 10 of these animals killed for necropsy 25 hours to 55 days after exposure, revealed the typical pathologic lesions. One animal killed after a 25-hour period had elapsed, and two others killed at the end of 4 months, were considered normal. One rabbit killed 6 days after a 30-minute exposure to 0.56 mg. per liter, revealed the usual evidences of damage, whereas another rabbit examined 4 months fter the same exposure was like the controls.

Exposure for 6 hours to atmospheres of 0.044 mg. per liter caused no deaths, though some evidence of liver and kidney damage was seen in three animals examined within 22 days following exposure.

One rabbit examined 18 hours following 41 hours of daily 6-hour exposures o 0.0245 mg. per liter had suffered considerable liver and kidney damage, with some evidence of an early fibrosing process in the emphysematous lungs. This concentration, although too low to cause death, is unsafe for prolonged xposure.

Exposure to concentrations of hydrogen fluoride above 1.5 mg. per lite for any period is dangerous for rabbits and guinea pigs.

Concentration of 1.0 mg. per liter and less caused no deaths in rabbit and guinea pigs exposed for as long as 30 minutes, although evidences of injury were definite.

Concentrations below 0.1 mg. per liter were tolerated by rabbits and guinea pigs for 5 hours without injury severe enough to produce death; 0.0245 mg. per liter caused no deaths in six animals exposed for 41 hours.

Machle and Kitzmiller (54) referred to available data that indicate that the lower limit of toxic concentrations of hydrogen fluoride in air lies between 0.03 and 0.0025 mg. per liter. The preceding paper in this study (2) demonstrated that the level lies below 0.0245 mg. per liter. Although no animals (of a group of 3 rabbits and 3 guinea pigs) died within year following 41 hours of daily 6-hour exposure at this level, the pathological findings on those sacrificed for necropsy revealed varying degrees of damage. Accordingly, the concentration of exposure atmospheres was lowered to 0.0152 mg. per liter. This concentration, though slightly irritating, was found to be tolerable and respirable, and it is therefore a level at which chronic human exposure could easily occur. Higher concentrations are sufficiently irritating to discourage prolonged human exposu

The effect of chronic ingestion of fluorine depends on the dose in proportion to body weight, the fluorine compound employed, the length of exposure, the nature and age of the individual, and the diet (3). An approximate relation between the symptoms of chronic poisoning and the dose for rat is given in table 4 from Roholm (3).

TABLE 4. - Relation between symptoms of chronic fluorine poisoning and dose for the rat

	Mg. fluorine per kg. body weight per day
Incipient with changes	1
Incipient bone changes, nephritis	5
First effects on general condition	10-15
Severe influence on general condition	20-25
Death in 1 or a few weeks	50-100

In man, 1 mg. per liter is the lowest concentration in drinking wate causing mottled teeth. The average weight of a child in the period duri which the permanent teeth calcify (from the first to about the tenth yea of life) is about 15 kg. If the daily intake of drinking water is put a 1 liter, then about 0.07 mg. fluorine per kg. per day can produce mottle teeth (3). In spontaneous osteosclerosis caused by cryolite, Roholm (3) approximated the fluorine intake at 0.2 to 1 mg. per kg. per day.

Table 5, giving lethal concentrations of hydrogen fluoride by inhal was prepared by Roholm (3) from data in the literature.

TABLE 5. - Lethal dose of hydrogen fluoride, in milligrams per liter

Death after 5 minutes respiration of 1.5 HF per liter - Rabbit and
Death after $\frac{1}{2}$ - $1\frac{1}{2}$ hours respiration of 0.6 HF per liter - guinea pig
Death after 1 day respiration of 0.03 HF per liter - Guinea pig
Tolerable for several minutes, but unpleasant, 0.026 HF per liter - Man

Permissible Limits of Hydrogen Fluoride in Air

The concentration of hydrogen fluoride stated as permissible in the air
s 3 parts per million (32). According to Shilen, Galloway, and Mellor (32),
owever, any statement of hydrogen fluoride in terms of parts per million at
mperatures below 90° C. is ambiguous. Below 90° C., hydrogen fluoride is
t the simple molecule H_1F_1 but is an associated molecule of one or several
ltiples of H_1F_1, sometimes stated as H_nF_n. Shilen and co-workers (32)
uote Treadwell and Hall as saying that the acid is bimolecular up to +40°,
ut above +80° it is monomolecular. Between these temperatures, both HF and
F_2 molecules are present. In dilute aqueous solutions, the simple mole-
ules preponderate, but in concentrated solutions at room temperature, there
s considerable H_2F_2. As the molecular weight of hydrogen fluoride below
0° or 100° C. is unknown, it is impossible to state toxic limits, but it is
ossible and correct to do so in terms of weight per unit volume of air, as
s done with the salts of hydrofluoric acid (32).

Machle and Kitzmiller (54) exposed animals 6 hours a day for 24 hours to
concentration of hydrogen fluoride of 0.0152 mg. per liter, or 15.2 mg. per
bic meter. The first animal died at the end of 160 hours of exposure.
hilen and co-workers state (32) it would be reasonable to believe that the
xposure when animals begin to die would be equally dangerous to humans.
ccording to Roholm (3), man seems to be more sensitive to fluorine com-
ounds than the experimental animals generally employed.

Referring to the experimental results of Machle and his co-workers,
hich showed that in a human experimental subject with a normal level of in-
ake of fluorine from dietary sources the urinary excretion and fecal output
ualed the intake within the limits of experimental error, Shilen and his co-
rkers (32) consider that any fluoride intake above 1.5 mg. per day is in ex-
ess of the normal that can be handled by the human body and evidently will
roduce some ill effect. They conclude, therefore, that any concentration
hich the ambiguous 3 parts per million of hydrogen fluoride may represent,
d its equivalent as sodium fluoride, is too high. Three parts per million
hydrogen fluoride is too high even if the molecule were H_1F_1, but as the
lecule is recognized as being associated as H_nF_n below 90° C. (194° F.),
arts per million of associated hydrogen fluoride raise the toxic limit
om 1 to an indefinite number when calculated in terms of sodium fluoride.

In the studies by Shilen and co-workers (32), the sodium fluoride equiva-
ent to the average of all atmospheric fluorine determinations was 0.996 mg.
er cm., but at the concentrations that gave this average, cases of respira-
ory ailments and dermatitis reported in greatest number.

Examination of the gases and fumes evolved during electrolysis of beryllium by Hyslop and co-workers (31) indicated that the fluoride content varied throughout the experiments between 0.07 and 5.0 mg. per cu. ft. The fumes consisted of a true vapor phase and a very fine smoke or dust. Analysis of the dust revealed beryllium fluoride, beryllium oxide, barium fluoride and sodium fluoride. With a single exception, all guinea pigs were killed within 90 minutes or less when exposed to the dust and fumes. The toxic effect apparently was due to the fluoride dust and fumes, for the elimination of any one metal from the melt did not lessen the toxicity appreciably. Analyses of the lungs of the animals that died or were sacrificed following exposure to the dust and fumes showed that the lung content of fluorine varied between 0 and 0.6 mg. per 10 gm. fresh tissues.

Machle and Evans reported in 1940 (55) results of a study begun in 193 of a group of workmen exposed principally to hydrogen fluoride, and to a lesser degree to the dusts of calcium fluoride, in the manufacture of hydrofluoric acid. As the hazardous nature of the operation was recognized and precautions were taken to prevent undue exposure at the higher levels of concentration, clinical examinations have revealed nothing significant. Determinations in 1940 of the fluorine content of the air in the center of the building gave negative results under normal operating conditions, but samples of air taken near equipment or while repairs were being made revealed 0.011 to 0.021 mg. fluorine per liter of air. The investigators concluded that exposure to these amounts eventually would result in clinical injury, as previous experiments showed that prolonged exposure to such concentrations would result in storage of fluorine in the bones.

PATHOLOGY OF FLUORINE POISONING

Roholm (3) summarizes the mode of action of fluorine as follows:

> In relatively large doses, fluorine causes a negative calcium balance, presumably by monopolizing the calcium of the organism; calcium fluoride is very slowly soluble. The calcioprive effect of fluorine cannot explain all the symptoms of the intoxication. The osteosclerosis is accompanied by increased mineral deposition, the tooth changes occur at such small doses that direct calcium deficiency is out of the question. Fluorine must be assumed to have a special effect on tissue. The effect on osseous and dental tissue appears universally by (1) the formation of an abnormally structured organic matrix, and (2) a calcification anomaly, whereby the mineral salts of the bone are precipitated irregularly and in discrete granules. Comparatively small doses seem to produce increased growth and increased calcification, comparatively large doses produce mostly atrophic processes and reduced calcification. The osseous effect of fluorine probably is the result of an influence on enzymatic processes connected with the precipitation of the mineral salts, stimulative or inhibitive according to circumstances. It is possible, but not probable, that the effect on the calcium metabolism is exerted via the parathyroid

glands. Fluorine affects several metabolic processes, and presumably the symptoms of intoxication have a complicated pathogenesis. There is a special relation between vitamin C and fluorine.

Fluorine compounds effect a large number of enzymatic processes (e. g. tissue respiration, blood coagulation, lactic acid formation in muscle, the splitting of starch by yeast). Generally the effect is inhibitory, but often a weak concentration of fluorine is stimulating, a stronger one inhibitory. Active fluorine compounds destroy protoplasm and arrest bacterial growth. · Yeast cells can become habituated to fluorine compounds. It is doubtful whether the quantity of fluorine absorbed under normal conditions is of any physiological significance. Experimentally, fluorine in quantities below the toxic limit has a stimulating effect on growth processes.

Machle and Kitzmiller (2) concluded that the effects were not entirely those of an irritant gas and suggested the possibility that the presence of altered fluorine-containing compounds contributed to the persistent blood changes and pathological findings. In later animal experiments with low concentrations of hydrogen fluoride, they (54) demonstrated that exposure to an atmosphere containing hydrogen fluoride, which caused no or little irritation, significantly lowered the erythrocite counts of exposed rabbits. Autopsies done on these animals 11 to 12 months after the initiation of the exposure period revealed evidences of damage to the lungs, livers, or kidneys of exposed animals, with a varying degree, location, and type of damage in the different animals. The lungs and liver were most severely and consistently affected, the one exposed monkey being exceptional in that respect. In all cases the lesions, though generally somewhat less severe, were like those seen in animals exposed to higher concentrations. The presence of typical lesions in the organs of these animals was especially significant in view of the low level of concentration of the exposure mixture, as was the persistence of pathological lesions many months after exposure had been discontinued.

FUNCTION OF FLUORINE COMPOUNDS IN PHYSIOLOGICAL PROCESSES

The finding of fluorine so widespread in plant and animal life has led to the suggestion that it has an essential physiological role in the animal kingdom. Roholm (3) stated that,

> It has never been demonstrated, nor is it probable, that fluorine in physiological doses is necessary to or has a beneficial effect on the development of teeth.

Gautier (56) claimed that fluorine had some bearing on the binding of phosphorus in the cell and that it contributed to the hardness and resistance of certain tissues (teeth, feathers) to various injurious chemicals. A number of investigators have stated that fluorine may be necessary for the proper development of the teeth and prevention of dental caries in man and animals, whereas others claim that animals fed on a diet low in fluorine appeared to be normal, although the bones and teeth contained extremely small quantities of fluorine (57).

McClure (58) has pointed out that, although the evidence obtained is not conclusive, there is the possibility of an optimum requirement of fluorine for the production and maintenance of normal dentine and enamel. He quotes Armstrong (59) to the effect that carious teeth appear to contain slightly less fluorine than normal teeth, but states that at present a requirement of fluorine for normal dental tissue and bone tissue or for any other body tissue or function has not been demonstrated.

Recent investigations by McClure (59) showed no relation of fracture perience to fluoride exposure of high-school boys and young adult males wh lived most of their lives in communities in which the fluorine content ran from none to 6.0 parts per million.

Dean (60) was the first to correlate statistically the incidence of dental caries with the fluorine content of the drinking water. The eviden from his thorough epidemiologic studies points strongly to the accuracy of the hypothesis that there is an inverse ratio of caries to fluorine found in drinking water.

Studies made by Dean (61) in 1938 revealed that in various areas of endemic fluorosis in the West and Middle West, there were more caries-free children than in communities where there was little or no fluorine in the drinking water. He suggested the control of dental caries by control of the water supply with a minimal threshold of safety of approximately 1 p. p. m. of fluorine.

In 1942, Knutson and Armstrong (62) obtained permission of parents in three small urban centers in Minnesota to make topical application of flu ride to the teeth of children aged 7 to 15 years. Data on the incidence dental caries in the permanent teeth of 289 treated children and 326 contr children for the year ended May 1943 have been presented and analyzed. During the first 8 weeks of the observation, the children in the treated group received 7 to 15 topical applications of sodium fluoride solution t the teeth in the left quadrants of the mouth. Analysis of the data indica that during the year of these observations approximately 40 percent fewer new carious teeth occurred in the treated than in the untreated teeth of t treated group of children. The relative reduction in the incidence of den caries in treated teeth was appreciably greater in the upper than in the lower teeth. There was no significant difference between the number of ne carious tooth surfaces in treated and untreated teeth which had been atta previously by caries. This finding indicates that the treatment was not effective in inhibiting renewed attacks by caries on teeth already attack The incidence of dental caries in teeth in the untreated mouth quadrants the treated group was similar to that in the comparable mouth quadrants of the control group. This finding indicates that for the first year follow treatment the caries inhibiting effect is local and limited to those teet to which the fluoride is topically applied.

Original from THE OHIO STATE UNIVERSITY

With the data available indicating the possibility that fluorine in non-xic doses might prevent the development of carious teeth, two long-range emonstrations to determine the practicability of mass protection against ental caries by adding fluroine to drinking water supplies have been initi-ted in two different sections of the United States.

The New York State Department of Health (63) recently began dental ex-inations of school children in Newburgh and Kingston, which have been elected as study and control areas, respectively. The purpose is to obtain, t the outset of the demonstration, a dental caries index that will serve as basis for comparison with the terminal figures at the end of 10 years, the ime that must elapse before the full benefits of the water treatment are ealized. This comparative analysis will make it possible to determine any mprovements that may have resulted from fluorination of the water. It is lso planned to make a pediatric investigation of a representative sample of he child population, including a general physical examination, urinalysis, nd X-rays of the long bones and centers of ossification. Procedures for hese examinations are now being prepared and, when they are completed, a ubstantial portion of the child population will be examined. All exami-ations will be made before the fluorine is added to the water supply.

Grand Rapids, Mich., has been chosen for another control experiment in ental caries under the auspices of the Michigan Department of Public Health, he United States Public Health Service, and the University of Michigan chool of Dentistry (64). The experiment will include the addition of 1 part er million of sodium fluoride to the municipal water supply to determine hether reductions can be made in dental caries. The plan to add the fluo-ine to the Grand Rapids water supply was approved by the City Commission. rand Rapids has been chosen because it has a stable population suitable for tudy over an extended period.

An interesting corroboration of the relationship to dental caries of ptimum concentrations of fluorine in drinking water is found in studies f caries in the teeth of 437 white selectees from 27 Illinois cities. bservations on these subjects were grouped by Deathridge (65) into those ho continuously used public water supplies of varied fluoride content hroughout life and those whose continuous exposure included the first 8 ears of life only. As these and other studies revealed that fluoride levels etween 0.0 and 1 p. p. m. markedly influenced the intensity of dental caries ttack and the resistence of the teeth to caries attack, a further study was ade of 2,026 white selectees living in 91 Illinois communities whose public ater supplies contained fluorides in different concentration. These waters ere grouped into the following three broad division: Fluoride-free (0.0-0.1 .p.m. of F), suboptimal fluoride (0.5-0.9 p.p.m.), and fluoride (1.0 p.p.m. nd more). Table 6 gives the total dental caries experience (omitting caries f the third molars) of the men.

TABLE 6. - Dental caries experience of selectees exposed to fluoride-free, 0.5-0.9, and 1.0 p.p.m. fluorides and over

Water supply	Teeth per man	
	Residence for entire life	Residence for at least first 8 years
Fluoride free	10.79	9.95
0.5-0.9 p.p.m. fluorides	7.88	8.87
1.0 p.p.m. fluorides and over	6.21	7.28

Individual susceptibility to fluorine is well-illustrated by a brothe and sister who had lived most of their lives in localities where the fluor in the water supply varied from none to 12 p.p.m. According to Linsman a McMurray (66), the brother joined the army at the age of 21. Admitted a y later to the hospital for treatment of a tumor of the eyelid, he was exami thoroughly. X-ray examination revealed an abnormal appearance of the bone the pelvis, entire spine, and the thoracic cage showed markedly increased density of the bony structures, so that the bones had almost the appearanc of chalk. Where this density had not completely obliterated structural definition in these bones, their trabeculae appeared thickened. Biopsy of the sternum showed grossly thickened trabeculae. The patient's condition was bad, and he died after the sternal wound, where the tissue was removed for biospy, became infected. Microscopic examination of the tissues showe essentially normal findings except in the urinary tract and osseous syste His condition may have been due to or aggravated by chronic fluroine pois

The patient was born and lived until 7 years of age in Spur, Tex., wh the fluorine content of the drinking water ranges up to 12 parts per milli From 7 to 9 years of age he lived in Post, Tex., where the drinking water contains 5.7 parts per million; then for 7 years his home was in Lubbock, Tex. (fluorine content of the water, 4.4 parts per million). He moved to Washington, D. C., but returned after 2 years to Lubbock, where he remain until he joined the Army at the age of 21 years.

The sister, who had spent all her life of 20 years in the same areas her brother, showed no signs of osteosclerosis on X-ray examination, alth she had a moderately severe degree of mottled enamel.

With reference to this case, Linsman and McMurray (66) state that enamel is undesirable cosmetically, but from the health standpoint there evidence that the affected teeth are less likely to become carious. Ost sclerosis due to fluroine, however, appears not to have any such virtue. may be dangerous as a cause of anemia. The sclerotic changes occur prim and most severely in the bones in which most of the blood is normally formed (66).

DETERMINATION OF CHRONIC FLUORINE POISONING

The usual methods of determining whether concentrations of fluorine which persons are being exposed may be harmful are examination of the te for mottled enamel, X-ray examination of the bones, analysis of the urin and determination of concentrations of fluorine in the air in industries where it may be present and in the food and drinking water consumed.

Examination of Teeth for Mottled Enamel

From results of studies on the toxicity of fluorine compounds, Smith and Leverton (67) concluded that, in concentrations far below those that interfered with the growth or general health of the animal, fluorine caused damage to the incisor teeth of the rat. All the compounds of fluorine studied, including the less soluble barium fluosilicate, cryolite, and calcium fluoride, left a mark characterized by the appearance of fine, lighter pigment lines on the incisor teeth when incorporated in the diet at a 0.0014 percent level (14 p.p.m.) or fed separately in daily doses of 0.2 mg. fluorine to young rats or fed at the level of 1 mg. per kg. of body weight. Fluorine, regardless of its source, interfered with normal tooth development at this level of feeding, but no discernible effect could be noted consistently when less than this amount was fed. The amount of fluorine required to cause initial damage to the rat incisors was so small that differences in solubility of the compounds were not a factor and no differences in toxicity of fluorine from the various compounds studied could be noted. The compounds studied were sodium, potassium, ammonium, and calcium fluorides; cryolite; and barium, sodium, and potassium fluosilicates.

According to McClure (58), very fine, closely spaced, brown striations may show on photographs of the teeth when the fluoride intake approaches 9 to 15 p.p.m. in food. Above these levels, the striations become progressively more pronounced, until they tend to become irregular patches at about 150 p.p.m. or more of fluorine in the intake, depending on the duration of the experiment. A chalky white appearance or lightening of the tooth pigment was not apparent to the naked eye with intakes equaling 9 to 15 p.p.m. of fluorine.

Dean, Sebrell, Breaux, and Elvolve (68) reported the appearance of fine striations at levels as low as 10.5 p.p.m. of added fluorine and later obtained indications of striations at levels of 8 to 10 p.p.m. Individual susceptibility is indicated by faint striations in photographs of some normal teeth, whereas photographs of teeth of animals exposed to low levels of fluorine ingestion showed apparently normal teeth.

Studies on the chemical composition of the teeth affected by chronic fluorosis reported by McClure (58) revealed that at levels of 0.012 to 0.025 percent of fluorine in the teeth, in general, the photographic appearance of the teeth is normal. At about 0.030 percent of fluorine the teeth may or may not show striations, but at 0.039 percent of fluorine the teeth become definitely striated.

The fluorine content of normal human tooth enamel and dentine has been determined by Bowes and Murray (69) as 0.02 to 0.03 percent. By spectroscopic analysis (68), enamel and dentine of teeth of individuals who had resided for 10 years previous to death in Colorado Springs and Cripple Creek, Colo. (where the drinking water contains approximately 2 p.p.m. fluorine), contained 0.065 percent and 0.112 percent fluorine, respectively.

135

Armstrong and Brekhus (60) reported the mean fluorine content of ena of sound permanent teeth to equal 0.0111 percent, with a range of 0.007 0.0167 percent. The fluorine in the enamel of mottled human teeth varied from 0.0245 to 0.0361 percent. In the dentine of four mottled human teeth analyzed, 0.0371 to 0.0425 percent fluorine was present. Normal dentine contains about 0.020 percent fluorine.

X-Ray Examination of the Bones

Chronic fluorine poisoning in man was first demonstrated by a peculia appearance of the bones in X-ray pictures of cryolite workers who were bei examined for silicosis (47). Roholm (3) found increased formation from b periosteum and endosteum (narrowed medullary cavity, periosteal deposits). Cancellous bone appeared densified and gave a diffuse, structureless shad Roentgenographic evidence of fluorosis in men exposed to fluorine in indus has been observed by other investigators. However, changes are visible ro genologically usually only after gross changes induced by excessive absorp of fluorine have occurred. Roentgenographic examination failed to reveal early macroscopic bone changes (observed at necropsy) that developed in al experimental animals fed by Largent and his co-workers (70) diets contai fluorine to the extent of 12 to 50 mg. per kg. per day for 16 to 92 days.

Analysis of the Urine

In an experiment on a human subject, Machle, Scott, and Largent (7 found that virtually all of the absorbed fluorine is excreted in the urin indicating that the urinary excretion is a convenient and accurate measur of the normal fluoride intake. Under normal dietary conditions, more tha 80 percent of the fluoride in the ingesta is absorbed, and all of this ab-sorbed fluoride is lost by way of urinary excretion; therefore, urinary ex-cretion can be used as a measure of fluoride ingestion. The equilibrium between intake and output is maintained when the daily intake and absorpt of fluoride does not exceed 1 mg. With higher levels of intake and absor-tion, the urinary fluoride excretion increases, the magnitude of excretio varying directly with the amounts absorbed. At high levels of intake and absorption, storage occurs even though there is increase in excretion. Machle, Scott, and Largent (72) found that storage occurred when the dail fluoride absorption was 2 mg. or more, the magnitude of storage varying directly with the absorption. As both urinary fluoride excretion and flu ride storage vary directly with absorption, they are correlated, and, the fore, urinary fluoride excretion may be used as a measure of fluoride st When 2 to 19 mg. fluoride were absorbed, about half was excreted; the oth half was stored. From clinical studies, analytical data on human necrops material, and from animal experimentation, Machle, Scott, and Largent inf (72) that storage occurs almost entirely in the bones.

Largent and Ferneau (1) tried this method to determine its usefulnes making preliminary appraisals of industrial exposure to fluorine compoun a magnesium foundry. The fluorine content of the urine samples showed th exposure was occurring in some foundry areas and not in others, and in s instances fluorine undoubtedly was being stored. However, they state (1) that no data are available to indicate the level of urinary excretion of fluorine that may be associated with injurious storage of fluorine in the bones.

DETERMINATION OF FLUORINE

There are a number of methods for the determination of fluorine in the air and in food and drink. The distillation, colorimetric, and titration methods have been described in detail by Jacobs (73). Most of the investigators referred to in this circular include their methods of analysis when reporting the results of their studies on fluorine poisoning. As most of the methods require too elaborate procedure to be included in this circular the reader is referred to the bibliography at the end.

Samples of dusty air and fume suspected of containing fluorine may be collected by the impinger or electrostatic precipitator. The samples may be analyzed or determined by various methods, such as that developed by Willard and Winter (74) and used, with various modifications, by many investigators.

Recently the development of a new test instrument has been announced (75). It is claimed that this tester affords rapid analysis of an atmosphere contaminated by hydrofluoric acid or sodium or calcium fluoride. The announcement states:

> The complete test kit weighs less than 5 pounds. No utilities or accessory equipment is needed to perform the test. Results may be obtained in less than 2 minutes by reference to calibration charts.
>
> Developed as an aid to safety engineers for the analysis of welding atmospheres and for use in light metal foundries, the instrument has proved of value for many other purposes. No technical knowledge is needed to conduct these tests if ordinary care in following instructions is used.
>
> The test consists of drawing the fluoride past a calibrated test paper, which instantly gives a change in color if fluorides are present. These test papers are guaranteed to be stable for 3 months.

PREVENTION OF FLUORINE POISONING

Plant Operating Precautions

Shilen and his co-workers (32) conclude from their studies of the hazard of fluorine poisoning in the manufacture of beryllium oxide that the atmospheric concentration of sodium fluoride should be reduced to 0.2 mg. (equivalent to 0.0952 mg. hydrogen fluoride and 0.0904 mg. fluorine), or less, per cubic meter to prevent sickness or injury to health.

General preventive measures more or less applicable to the smelting a petroleum industries are general ventilation and as complete enclosure as possible of all processes to prevent escape of dust and fumes into the atm phere breathed by the workers. Many processes require special spot ventil tion or other special measures.

At the Teterboro, N. J., Eclipse-Pioneer metal-casting division of th Bendix Aviation Corporation (76), which turns out magnesium, aluminum, bronze, and brass castings for the armed forces, a battery of 2 dozen "iro lung" ventilators, each capable of "inhaling" 30,000 to 45,000 cubic feet of air per minute, has enabled the division to step-up production of these castings 20 times above prewar levels. These iron lungs are used to remov smoke and fumes from the scene of metal-pouring operations, which previous had spread to all parts of the foundry, interfering with the efficiency of workers in core-making, molding, heat-treating, and other departments. Th device is a power ventilator designed to discharge impure air so high abov the roof that it cannot re-enter buildings through open windows, doors, o idle ventilators. It is constructed with an unobstructed "throat," said t be leakproof (76). When its fan is operating and dampers are open, rain snow are expelled by the force of the discharged air stream. Operating motors are geared to individual plant or foundry conditions.

In the Eclipse-Pioneer foundries (76), this type of ventilation is in stalled on roofs over locations most continuously affected by smoke and f from melting and pouring and shake-out jobs. The iron-lung ventilator ha been tested in constant operation and found satisfactory.

To prevent contamination of the soil and vegetation around aluminum plants, the fluoride-containing materials should be collected by a mechan cal ventilating system and discharged through tall stacks or passed throu scrubbers to remove the contaminant. All pots should be ventilated prop to prevent the escape of fumes when the doors are opened. Each pot shoul be provided with positive volume control and separate shut-off damper. A parts of the duct work of the ventilation system should be kept clean. A system of recovering the solids in the stack discharge might be profitab and would help control the nuisance to the neighborhood. High stacks are desirable for dispersing unrecovered material.

In some hydrogen fluoride alkylation plants, measures are incorporat in plant design to minimize as much as possible the hazard of accidental lease of acid into the atmosphere. In the plant of the Universal Oil Pr Co. (38) an exhauster system discharges to a caustic wash tower for neut zation of hydrogen fluoride vapors before release to the atmosphere. Th exhaust release is located away from the plant area, so that if the caus system fails, the hazard to persons in that area will be minimized. Foll is a description by Benson (38) of neutralization and disposal of acid s in the plant:

> Quick neutralization and disposal are necessary when there are accidental spills of acid in the plant area caused by failure to evacuate properly, by failure of the seals on pumps,

contactors, or valves, by rupture of lines or vessels, or by any other untoward incident. For this purpose, the acid-carrying equipment is located in a pit containing a 2-foot depth of limestone, with the pit wall extending about 18 inches above the base level to serve as a retaining wall. This wall or curb serves also as a boundary for the restricted acid area. The limestone neutralizes the acid which it contacts so that its spread to the surrounding area is avoided; and contamination by seepage of water through the soil is prevented, as well as corrosion of underground piping and foundation work.

Limestone that has reacted with the acid is removed, and new limestone is laid down after the area is thoroughly washed with water. The spent limestone is deposited in an isolated location away from any processing equipment, where it is allowed to hydrolyze slowly.

An auxiliary limestone pit is provided outside the acid area into which water and/or acid following major spills may drain from the acid-area pit for further treatment.

An acid-area pit which does not contain limestone may also be used. In this case water washing is done immediately following the spills; and dilute acid solution is then neutralized in an auxiliary pit containing limewater, and the acid is precipitated out. The precipitate is subsequently removed and buried, the liquid being discharged through the sewer after it becomes neutral.

Disposal of spent acid should receive continued attention so as to avoid contamination of drinking water and pollution of rivers and streams.

* * * * * * * * * * *

Spent buaxite from the bauxite treaters, contaminated limestone from the neutralizing pit, precipitated calcium fluoride, or waste materials which cannot be easily and completely decontaminated must be disposed of in a safe manner and in a place where they will not constitute either a hazard or a nuisance. These materials should be dumped into a pit over the bottom of which has been placed a layer of lime. Lime should also be spread on top of the dumped material so that any free fluorine remaining in it will be converted to stable calcium fluoride. Flammable materials which may contain HF should be burned.

Identification of all piping in which there is or may be hydrogen fluoride should be in accordance with the color scheme of the A.S.A. Code. This Code stipulates yellow as the color for hazardous materials. Lines containing other materials are to be painted in accordance with the respective colors specified.

Acid spills on floors, stairways, and platforms should be immediately neutralized with soda ash. Small pans containing this chemical should be provided to catch drippings.

All equipment which has been in acid service should be thoroughly neutralized before it is turned over to shop or warehouse, or placed elsewhere for subsequent disposal where it may be handled with the bare hands. A strong soda ash solution is recommended as a neutralizer. Shop repair and maintenance men, warehouse men, and others should be warned of the danger of HF so that they will be on the alert for any indication of acid.

According to Gerhold and his co-workers (77), the problems of the desi engineer were simplified somewhat by recent developments in instruments, su as wide-range level controls, torque-tube transmission, aneroid differentia instruments, and remote electric flow meters. For safety, all instruments that contain hydrogen fluoride are mounted locally, and either air or electricity are employed for transmission to or remote control from the main instrument board.

In its pamphlet on Recommended Practice for Unloading Anhydrous Hydrofluoric Acid From Tank Cars, the Manufacturing Chemist's Association of the United States (78) suggests that workmen unloading cylinders should be full informed of the dangers incident to improper handling. The departmental supervision should require rigid enforcement of all safety rules and precautions.

Protection to workmen unloading cylinders containing anhydrous hydrofluoric acid should include wearing long-gauntlet neoprene gloves; rubber shoes, or preferably rubber boots; protective apron, rubber coat, or rubbe overalls; and proper goggles or face shields. It is essential that this equipment be maintained in good condition. Gloves and shoes should be inspected frequently to insure against defects that might result in leaks. liquid acid gets on gloves, they should be washed immediately. Transparen plastic face shields or goggles should be worn when operating valves or wh making any close observations that, in the case of a failure, might cause acid to be discharged into the face. In breaking connections on pipe line or other acid equipment, the operator should be protected by a full-vision acid hood, rubber coat, boots, and gloves.

Contact of the acid or its fumes with goggles or glass spectacles wil etch the lenses. When this occurs, they should be replaced immediately. Application of a thin film of vaseline to both inside and outside surfaces of the glass will retard such etching. It is advisable that goggle lenses be made of a material impervious to the acid, such as methyl-methacrylate plastic.

Personal Protective Devices

Personal protective devices include protective clothing and respirat

The various types of jobs in the acid area of HF alkylation plants re- iring specific protective wearing apparel are classified by Benson (38) s A, routine operations; B, plant alterations; C, maintenance; and, D, ergency or hazardous work. He discusses the clothing required for each pe of job as follows:

Class A

Class A clothing, which consists of neoprene-soled work shoes, a rubber cap, a wide-angle face shield, and neoprene gauntlet gloves, is worn only when making a visual observation of the plant under normal conditions. Any person entering the acid area must wear this equipment; his gloves must be on and his face shield down when he crosses the restricted-area boundary line. The shoes protect his feet against acid that may have condensed on the ground or on the stairways, platforms, and runways. The cap offers protection against acid dropping from above. The gloves are extremely essential, because any surface touched may have an acid film; or in performance of various duties there may be slight discharges of acid vapor, which may come into contact with the unprotected hands by way of tools and other objects that may be handled. The wide-angle feature of the face shield is a protection also to the ears.

Class B

When changes are to be made in plant operation, Class B clothing should be worn. This clothing includes a rubber or neoprene acid-proof coat, together with the Class A apparel mentioned above; i.e., neoprene-soled work shoes, rubber cap, face shield, and neoprene gauntlet gloves. The purpose of the coat is to provide protection for the arms and body of the workman. The possibility of pump and packing failures, for instance, when making adjustments to operating conditions prompts this recommendation for additional protection.

Class C

Clothing of this classification is recommended whenever acid equipment is to be opened up for inspection or repair. Rubber boots and neoprene acid-proof garments, which include a coat, overalls, guantlet gloves, and a hood, are worn. Rubber apparel may be substituted if neoprene is not available. Class C clothing protects the wearer against the discharge of acid or acid vapor which may not have been previously drained or evacuated from the inside surface of the equipment.

Class D

This class of protective apparel has been specifically designed for work in acid vapor atmospheres where complete protection might be needed, as in the case of repairing an extremely large leak, or of working where the concentration is higher than that which could be handled by a cannister mask. The clothing consists of a neoprene coverall suit provided with the novel feature of admitting a compressed fresh-air supply to the body as well as the headpiece. Through the admission of air to the body portion, the wearer is enabled to move about in comfort; in addition, the air prevents acid vapor from entering the suit because of the air passing out. The constant rush of air blowing out around the neck and other openings makes it possible for the wearer to work safely in any concentration of acid vapor-air mixtures. This feature also is a safeguard in case the suit is torn; the vapors in this instance as well could not enter.

The air supply for this suit is taken off the air line referred to previously as having been installed for that purpose. Operators and others should be required to practice donning the suit so that there will be no loss of time when an occasion arises when the suit should be used. The alkylation plant should be provided with a large and a small suit, and both suits should be readily available at all times and regularly examined for condition.

Safety apparel should be thoroughly washed with water before it is laid away. A suit can be conveniently washed before the wearer takes it off.

A company manufacturing beryllium oxide from beryl (32) provided respirators for protection against dust and fumes. It was observed, however, th the supervision necessary to the proper wearing and use of these protective devices was lacking.

It should be emphasized that the use of respiratory protective devices as control procedure requires as much consideration as any other control method (79). Air-line respirators or mechanical filter fume respirators, approved for protection against the inhalation of solid dispersoids or particulate matter formed by the condensation of vapors such as those from heated metals and other substances, would afford protection in plants wher the dust and fume contain fluorine.

Although choice of a suitable respirator is important, other considerations are equally so (80). Obviously, if a respirator is to do the job f which it is chosen, it must be worn properly and be kept in good condition. Frequently, persons do not know how to wear a respirator, and if it is not clean, it is unpleasant to wear. Better protection and fuller cooperation doubtless would be obtained if the respiratory protective devices were kep under the supervision of a responsible person who would see to it that the wearers are properly instructed in their use, that the devices are cleaned and disinfected regularly, and that they are maintained in first-class condition.

Physical Examination

X-ray examinations of workers before they are assigned to operations that may expose them to a possible fluorine poisoning hazard, to eliminate those with abnormal bone and joint conditions, with subsequent annual X-ray examinations are routine in some plants.

In connection with X-ray examinations attention is called again to the statement by Largent, Machle, and Ferneau (70) that roentgenographic examination failed to reveal early bone changes in animals with greatly increased fluoride content of the skeleton.

An additional safety measure would be pre-employment analysis of the urine for fluoride content with routine analyses every 6 months, as advised by Benson (40). If normal content is known, subsequent analyses would show whether any fluoride was being absorbed.

TREATMENT OF FLUORINE POISONING

The most frequently experienced acute ill effect of exposure to fluorine is hydrofluoric acid burns that may occur among workers making, transporting, and using anhydrous hydrofluoric acid and its aqueous solutions. The following treatment for such burns is recommended by the Division of Industrial Hygiene of the National Institute of Health(78):

> The burn from the aqueous solution of hydrofluoric acid may not be felt until an hour or more after exposure, but the burn from anhydrous hydrofluoric acid is felt immediately. The length of time elapsing after exposure, before the burn is felt, depends upon the concentration of the acid. If the exposure is for a short time, a minute or so, to a low concentration, or momentarily to a high concentration, a blanched white slightly edematous macerated appearing area of skin is seen. If the exposure is for a long period to a low concentration or for a minute or so to a high concentration, the skin appears red and turns to a greyish purple with considerable edema and tension.
>
> Burns from hydrofluoric acid may be placed into three classes:
>
> (1) Those resulting from concentrations up to 20 percent which manifest themselves several hours after exposure by a deep seated reaction.
>
> (2) Those from concentrations of approximately 20 to 50 percent in which the latent period is shorter.
>
> (3) Those from concentrations of approximately 60 percent to anhydrous hydrofluoric acid in which the burn is felt shortly after or immediately upon exposure.

Workers who have had hydrofluoric acid in contact with the skin should immediately step under a drenching shower and immediately remove all the clothes, and then receive medical attention as quickly as possible. If the eye is affected it should be copiously flushed with water as soon as possible. If it is anticipated that 20 minutes or longer will elapse before medical attention is received, then apply to the burned area a wet dressing consisting of 20 percent magnesium oxide in glycerin or a magnesium oxide sulfate paste.

If it is known that the acid only touched a localized site, such as the hand or arm, it should be immersed in running water for three to four hours, the triple dyes applied and over this a dressing with magnesium oxide paste.

Calcium gluconate 10 percent should be injected as soon as possible into, under, and around the burn to prevent spreading and to relieve pain.

The periphery and base of small ulcers should be injected with the gluconate solution and the base curetted and treated with the triple dyes.

If the nails are painful, swollen and discolored, a local anesthetic should be injected at a considerable distance from the inflamed area and the nail opened down to the matrix. This relieves the pressure and pain. A portion of the nail is cut away, calcium gluconate injected into the matrix, the triple dye applied, and the part dressed with the magnesium oxide paste dressing.

Nasal ulceration should be treated by gently wiping away the crusts and generally keeping the nasal mucosa clean. It can be prevented or kept from progressing by proper exhaust vents over operations, by wearing suitable respirators, and by the insertion of petroleum jelly into the nose.

If the eye is burned, it should be washed every five minutes with physiologic saline solution until the physician arrives. Severe pain may be alleviated by instilling a drop of local anesthetic into the eye (avoid cocaine if possible). If the cornea is also affected and there is photophobia, a drop of a 1 percent solution of atropine may also be instilled. It is advisable to put an occlusion dressing over the eye which may be removed later on and followed by dark glasses.

Acute poisoning as a result of swallowing fluoride compounds or inhal
and swallowing dust containing fluorides may be treated by gastric lavage
calcium chloride followed by absorbents such as charcoal (46).

Apparently there is no treatment for chronic fluorine poisoning exce
avoidance of exposure to the dust and vapors. From his examination of cr
lite workers who had not worked in the industry for a long time, Roholm (
concluded that there was a possibility that the effect upon the bones mig
disappear to some extent. So far as known, there is no treatment that wi
restore the mottled enamel of teeth.

Barach (81) states that the pathologic condition found after inhalation
f irritating gases (among which he mentions hydrogen fluoride and fluorine)
s that of intense congestion and inflammatory edema followed by a cellular
udate with a varying amount of necrosis of the lining membrane of the re-
piratory passage. The Journal of the American Medical Association (81)
escribes his method of treatment as follows:

> Barach and his colleagues have developed an apparatus for administration of positive pressure in cases of pulmonary edema produced by gas poisoning. Inhalation of oxygen and helium-oxygen mixtures under positive pressure is of value in the treatment of obstructive dyspnea and acute pulmonary edema. The only contraindication to positive pressure respiration is the presence of shock. The employment of positive pressure respiration in cases of industrial gas poisoning indicates that endema of the lungs may be prevented and, when it has occurred, tends to respond to the administration of positive pressure in a far more successful way than with any hitherto suggested procedure. A pressure of 3 to 6 cm. of water is recommended. Prevention of pulmonary edema after exposure to an irritant gas is accomplished by inhalation of air or oxygen against positive pressure for 2 to 3 hours and subsequent trial periods of freedom from positive pressure to determine whether or not edema intervenes. Since pulmonary edema may take place as late as twelve hours after exposure, means for providing positive pressure should be at hand. The inhalation of a spray containing 0.5 to 1 percent epinephrine is helpful in cases in which bronchial spasm is present, and inhalation of 0.5 to 1.0 cc. of 1 percent neosynephrine is suggested to increase the lumen of the tracheobronchial tree by vasoconstriction. Immediate administration of positive pressure is suggested in disasters in which phosgene or other gases are liberated.

SUMMARY

The increasing use of hydrogen fluoride in industry, especially in metal-
rgical processes and petroleum refining, and complaints of injury to live-
tock and vegetation from the gases and dust discharged by some of the plants
ave aroused interest in its toxicity. A review of the literature on this
bject therefore seems opportune.

The name "fluorine" is derived from the characteristic property of fluo-
ine minerals of acting as fluxes (promoting the fusion of other minerals).

Fluorine is prevalent in soil, rocks, sea water and springs, and in the
nes, teeth, blood, and milk of animals.

The principal fluorine-bearing minerals that are used in large-scale
dustrial processes are fluorspar (CaF_2), cryolite (Na_3AlF_6), apatite
$3Ca_2(PO_4)_2.CaF_2$), and sedimentary phosphate rock.

The largest users of fluorspar are the steel, hydrofluoric acid, and glass and enamel industries, and it is employed extensively in insecticides especially in spraying fruits.

Free fluorine plays no part in toxicology, as it immediately reacts wi water, forming hydrogen fluoride.

Hydrogen fluoride is a colorless gas (formula HF, molecular weight 20. that condenses to a colorless liquid at 19.5° C. It has a marked affinity water, forms a fog with water vapor in air, and when dissolved in water yie a constant boiling mixture containing 38.2 percent HF, which distills at 110.8° C. at 732 mm. mercury, and having a specific gravity 1.138 at 20° C.

Hydrofluoric acid (HF in aqueous solution) is a colorless, fuming, mob corrosive liquid.

The physical properties of HF give it considerable technical advantage over many other substances for similar large-scale industrial use. Its boi ing point is near room temperature, and therefore it can be handled either a liquid or a gas without the use of high temperatures or pressures. It ha a very low surface tension; this, with the high density, enables the liqui to mix readily with hydrocarbons by agitation but allows the mixture to separate readily by settling.

Fluorine minerals have been known since the Middle Ages. The first attempt to etch glass by the vapors was made in 1670. The aqueous solutio of HF was produced in 1771. The presence of fluorine in a fossil tooth was demonstrated in 1803. The corrosive effect of the acid on the skin was decribed in 1809. The wide distribution of fluorine in nature was determi in 1846.

It is only within comparatively recent years that the serious nature fluroine poisoning has been realized.

The first indication of fluorine poisoning from industrial plants was the appearance of a peculiar disease in animals pastured close to smelters and other fluorine-using plants.

Probably the most spectacular instance of the ill effects of fumes di charged from factories was the fog disaster in the Meuse Valley in Belgiu December 3-5, 1930, which caused several thousand cases of acute pulmonary attack and 60 deaths. The illnesses and deaths have been attributed to fl rine compounds in the gases discharged from industrial plants in the valle 15 of which handled raw materials containing fluorine or added fluorine compounds to the raw materials.

A peculiar condition of the teeth, called "mottled enamel" in the Uni States, mentioned in the early literature as occurring in various parts of world, was not definitely connected with exposure to fluroine until 1916, the results of a study of the disease in Colorado were reported. The dise is endemic in certain States of tho United States, in South America, Chin olland, Spain, Italy, Mexico, South Africa, Bahama, and the Cape Verde slands. The disease did not occur where the fluorine content of the soil as lower than 20 mg. in 100 g.

Chronic fluroine poisoning in human beings was discovered in 1931 among orkers in a cryolite factory who were being examined for silicosis. The -ray revealed a peculiar sclerotic bone affection. In pronounced cases the ffection had attacked all the bones of the organism, particularly the ertebral column and pelvis.

In addition to the uses of fluorine in large-scale industrial operations, l technical processes have been listed as possible sources of poisoning. One tate Health Department has listed 39 occupations in which contact with fluo-ine and its compounds is indicated.

The reduction of aluminum may be accompanied by a hazard of poisoning by luorides inside the plant as well as outside through pollution of the atmos-here by the gases and fumes discharged from the stacks.

Aluminum is made by the electrolysis of alumina (Al_2O_3) dissolved in a ath of molten cryolite. The principle of the method used in the decomposi-ion by the heat of the electric current of the cryolite and its combination th the silica of the carbon of the electric arc to form silicon fluoride as, which is transformed almost immediately into fluosilicic and hydrofluoric cid. The hydrofluoric acid thus produced may affect not only the plant ersonnel but persons within a radius of several miles.

The slight etching of the glass windows in aluminum reduction works re-eals the liberation of fluroine; this same incidence of fluorine in the tmosphere may be noticed sometimes in the windows in the vicinity of aluminum lants.

The principal fluorine exposure in aluminum plants is in working around e pots - adding the charge, changing electrode connections, renewals, and milar operations that require opening the doors of the pots for a short eriod at regular intervals. When the doors are opened wide, the hot cur-ents generated by the hot bath carry most of the fumes out into the room, ere the workers are exposed unless each pot is properly ventilated.

In plants with mechanical ventilating systems, large quantities of fumes re discharged from the roof monitors and from the stacks. Most of these mes descend to ground level and are deposited on the vegetation within a rtain radius of the plant, in accordance with the prevailing direction of e air currents, and constitute a hazard to animals that eat the vegetation.

The recently increased production and use of beryllium has led to oppor-nities for exposure to fumes and dust in virtually all stages of beryllium duction and in the manufacture of beryllium oxide and its use in beryllium loys.

Investigations of the toxicity of beryllium led to the conclusion that the substance itself is not toxic and that the illnesses of workers expose to the dust and fumes produced during the extraction of beryllium are due t various fluorides, including beryllium oxyfluoride. The latter, when smelt produce vapors that provoke serious pulmonary affections.

In one study, the fluoride content of gases and fumes evolved during electrolysis of beryllium varied between 0.07 and 5.0 mg. per cubic foot. With a single exception, all guinea pigs were killed within 90 minutes or less when exposed to the dust and fumes.

Hydrogen fluoride poisoning also may occur in magnesium founding, in which a large group of soluble fluoride salts are used as inhibitors. The chief exposures to fluorides are during the shakeout, molding, melting, an core spraying operations. The concentrations of fluoride found during thes operations in a magnesium foundry were, in milligrams per 10 cubic meters, 9.4 to 180 (average 87.7), 4.4 to 106 (average 18.8), 7.4 to 17.6 (average 12.6), and 5.8 to 9.4 (average 7.0), respectively.

Hydrogen fluoride is being used advantageously as a catalyst in the alkylation of olefins to produce high-octane motor fuel. The first hydrog fluoride alkylation plant began operation in December 1942, and several mo were built during 1943. The principal sources of poisoning in these plan are accidental spills of acid in the plant area due to failure to evacuate properly, failure of seals on pumps, contactors, or valves, or rupture of lines or vessels. Burns may result from handling tools that may have been in contact with the acid. There is a possibility of pump and packing failu where adjustments are made to operating conditions. Repairmen may be expo to high concentrations of hydrogen fluoride while repairing an extremely l leak. It is almost impossible for anyone consciously to inhale enough hyd fluoride to harm himself seriously, as the acid gas is too pungent for voluntary inhalation.

In addition to the industries mentioned, there are others in which a possible fluorine poisoning hazard exists, such as welding, pickling metal manufacturing hydrofluoric acid and fertilizers, and many other less impor occupations from the standpoint of number of persons exposed.

Fluorine poisoning may be acute or chronic. Acute symptoms occur mos frequently from ingestion by mouth of fluorine compounds. Symptoms in su lethal cases are acute gastroenteritis with sudden nausea and vomiting, abdominal burning and diarrhea; fatal cases may develop alternating convul sions or spasms and paresis (slight paralysis), with general weakness and sluggishness between the spasms, fall of blood pressure, general collapse, sometimes with grayish-blue cyanosis, dyspnea, and arrest of respiration of the heart. Death may occur in a few minutes or may be delayed for 10 hours or longer.

Changes in the teeth are among the most easily recognized symptoms chronic poisoning. Fluorine has a detrimental effect upon growth of the teeth, and the parts of a tooth calcifying during the period of fluorine intake will acquire permanent defects.

The most significant symptoms of chronic fluorine poisoning from the health standpoint are the changes in the osseous system. After absorption, fluorine is stored in the body, especially in the bones and teeth. Ability to move the spinal column spontaneously often is restricted and parallels osseous changes. The most common clinical symptoms of chronic fluorine poisoning are dyspeptic phenomena, shortness of breath, and rheumatic attacks. Loss of appetite, nausea, vomiting, and heartburn were experienced by 80.9 percent of the workers and circulatory and respiratory symptoms by 51 percent in a cryolite factory where they were exposed to dust. Some of the workers complained of continuous constipation.

Inhalation of an atmosphere containing 1.0 to 1.5 mg. hydrogen fluoride per liter kills a significant proportion of animals exposed for 5 minutes or more. No deaths occurred in two groups of animals exposed for 5 minutes to 0.79 and 0.50 mg. per liter, respectively, but 7 of 10 of these animals killed for necropsy 25 hours to 55 days after exposure revealed the typical pathologic lesions. One rabbit exposed to 0.0245 mg. per liter 6 hours daily for 41 hours had suffered considerable liver and kidney damage, with some evidence of an early fibrosing process in the emphysematous lungs. Although this concentration is too low to cause death, it is unsafe for prolonged exposure. A concentration of 0.0152 was slightly irritating but tolerable and respirable; however, it is a level at which chronic human exposure may occur, as higher concentrations are irritating enough to discourage prolonged human exposure.

Some investigators believe that the generally accepted permissible concentration in air of 3 p.p.m. is too high and that, as the molecular weight of hydrogen fluoride below 90° to 100° C. is unknown, it is impossible to state toxic limits except in terms of weight per unit volume of air, as is done with the salts of hydrofluoric acid, and that any fluoride intake above 1.5 mg. per day is in excess of the normal that can be handled by the human body.

The principal pathologic effects from fluorine poisoning are blood change, damage to the lungs, liver, and kidneys, and storage of fluorine in the bones. The lungs and liver are most severely and consistently affected.

Its widespread occurrence in plant and animal life has led to the suggestion that fluorine plays an essential physiological role in the animal kingdom. A number of investigators state that fluorine may be necessary to the proper development of the teeth and the prevention of dental caries in man and animals. Others claim that animals fed on a diet low in fluorine appeared to be normal, although the bones and teeth contained extremely small quantities of fluorine.

Comparison of the occurrence of dental caries among residents in communities where the content of the drinking water is below 1 p.p.m. with the occurrence among those living where the drinking water contains more than 1 p.p.m. indicates that the latter have fewer caries. This is confirmed by examination of selective service men. Those who had resided all their lives where drinking water was fluorine-free had 10.79 carious teeth each; where the drinking water contained 0.5 to 0.9 p.p.m. of fluorine, the men had 7.8 carious teeth each; and where the drinking water contained 1.0 or more p.p. of fluorine, the number of carious teeth per man was 6.21. Those who had resided for at least the first 8 years where the drinking water was fluori free had 9.95 carious teeth per man; where the drinking water contained 0.5 to 0.9 p.p.m. fluorine the number of carious teeth per man was 8.87; and where the drinking water contained 1.0 p.p.m. or more fluorine the number o carious teeth per man was 7.28.

Individual susceptibility is illustrated by a brother and sister, livi for 20 years under the same conditions of exposure to fluorine in drinking water. X-ray examination of the brother showed considerable sclerotic bone changes and anemia was present but no mottling of the teeth, while the sist teeth were mottled but she was not anemic and X-ray examination revealed no change in her bones. The brother died from infection of the wound where tissue was removed for biopsy (examination of living tissue for diagnosis). The sclerotic changes occur primarily and most severely in the bones in whi most of the blood is formed normally.

The usual methods of determining chronic fluorine poisoning are examination of the teeth for mottled enamel, X-ray examination of the bones, ana sis of the urine, and determination of concentration of fluorine in the ai industries where it may be present and in the food and drinking water.

Photographs of the teeth may reveal very fine, closely spaced, brown striations when the fluoride intake approaches 9 to 15 p.p.m. in food. In studies on the chemical composition of the teeth affected by chronic fluorosis the photographic appearance of the teeth with a content of 0.012 to 0.025 percent of fluorine was normal. With a content of 0.030 percent flu rine, the teeth may or may not show striations, but with 0.039 percent of fluorine, the teeth become definitely striated. Normal dentine contains about 0.020 percent fluorine.

Chronic fluorine poisoning was first demonstrated in man by a peculia appearance of the bones of cryolite workers being examined for silicosis. Bone changes are visible roentgenologically usually only after gross chang induced by excessive absorption of fluorine have occurred.

Under normal dietary conditions more than 80 percent of the fluoride the ingesta is absorbed and lost by way of urinary excretion. The equilib between intake and output is maintained when the daily intake and absorpti of fluoride does not exceed 1 mg. At high levels of intake and absorption storage occurs even when excretion is increased. When 2 to 19 mg. fluori were absorbed about half was excreted; the other half was stored. As urin fluoride excretion and fluoride storage vary directly with absorption, the are correlated and urinary fluoride excretion may be used as a measure of fluoride storage. However, no data are available to indicate the level of urinary excretion of fluorine that may be associated with injurious storag of fluorine in the bones.

There are numerous methods for determining fluorine in the air and in food and drink. Most of the investigators referred to in this circular include the methods of analysis used in their studies on fluorine poisoning. Samples of dusty air and fume suspected of containing fluorine may be collected by the impinger or electrostatic precipitator and analyzed by various methods, the most frequently used being modifications of the Willard and Winter method. Recently a new test instrument has been announced with the claim that its use requires no technical knowledge and affords a rapid analysis of an atmosphere contaminated by hydrofluoric acid or sodium or calcium fluoride.

General measures to prevent poisoning more or less applicable to the smelting and petroleum industries are general ventilation and as complete enclosure as possible of all processes to prevent escape of dust and fumes into the atmosphere breathed by the workers. Many processes require spot ventilation. Quick neutralization and disposal of acid spills are necessary in plants using fluorine. All equipment that has been in acid service should be neutralized thoroughly before it is placed elsewhere for subsequent disposal, where it may be handled with the bare hands. Workmen should be fully informed of the dangers of handling fluorine containers.

Personal protection includes wearing respirators, rubber gloves, shoes, apron, coat, or overalls, and proper goggles or face shields when necessary. Goggle lenses should be made of material impervious to the acid as the acid will etch glass.

Pre-employment X-ray examination and annual X-ray examinations subsequent to employment are routine in many plants. An additional safety measure would be pre-employment analysis of the urine with routine analysis every 6 months. If the normal fluorine content of the urine is known, subsequent analyses would show whether fluorine is being absorbed.

The most frequently experienced ill effects of exposure to fluorine is burns. Workers whose skin is in contact with hydrofluoric acid should immediately wash the place with water and receive medical attention as soon as possible. If the eye is burned, it should be washed every 5 minutes with physiologic saline solution until the doctor arrives.

Acute poisoning as a result of swallowing fluoride compounds or inhaling and swallowing dust containing fluorides may be treated by gastric lavage with calcium chloride followed by absorbents such as charcoal.

Apparently there is no treatment for chronic fluorine poisoning except avoidance of exposure to dust and vapors containing fluorine. There is a possibility that the effect on the bones might disappear to some extent with removal from exposure to fluorine. There is no known treatment that will restore mottled enamel teeth to normal.

Inhalation of oxygen and helium-oxygen mixtures under positive pressure is said to be of value in the treatment of the obstructive dyspnea and acute pulmonary edema caused by inhalation of irritating gases, including fluorine.

BIBLIOGRAPHY

1. LARGENT, E. J., and FERNEAU, I. F. Exposure to Fluorides in Magnesium Founding. Jour. Indust. Hyg. and Toxicol., vol. 26, 1944, pp. 113-1

2. MACHLE, WILLARD, THAMANN, FREDERICK, KITZMILLER, KARL, and CHOLAK, JAC The Effects of the Inhalation of Hydrogen Fluroide: 1. The Response Following Exposure to High Concentrations. Jour. Indust. Hyg., vol. 16, 1934, pp. 129-145.

3. ROHOLM, KAJ. Fluorine Intoxication - A Clinical-Hygienic Study With A Review of the Literature and Some Experimental Investigations. Lond 1937, 364 pp.

4. JOHNSTONE, RUTHERFORD T. Occupational Diseases. 1941, pp. 146-153.

5. MELLOR, J. W. Modern Inorganic Chemistry. 1918, pp. 275-281.

6. DAVIS, H. W., and TROUGHT, M. E. Flurospar and Cryolite. Bureau of Mines Minerals Yearbook, 1940, pp. 1333-1352.

7. SIMONS, J. H. Hydrogen Fluoride - The Catalyst. Petroleum Refiner, v 22, 1943, no. 6, pp. 83-89; no. 7, pp. 83-87.

8. GERHOLD, C. G., IVERSON, J. O., NEBECK, H. J., and NEWMAN, R. J. The Development of the Hydrogen Fluoride Alkylation Process. Pet. Ref., vol. 22, 1943, pp. R.146-R.155.

9. DeEDS, FLOYD. Chronic Fluorine Intoxication, A Review. Medicine, vol 12, 1933, pp. 1-60.

10. OST, H. Der Kampf gegen schädliche Industriegase. (The Fight Against Harmful Industrial Gases.) Zeitschrift für angewandte Chemie, vol. 1907, pp. 1689-1693.

11. CAMERON, C. A. On the Toxicity of Silicon Fluoride. Dublin Jour. Med Sci., vol. 83, 1887, p. 20.

12. VAN LEEUWEN, W. STORM. Die Nebelkatastrophe in Industriegebiet südli von Lüttich. (The Fog Catastrophy in the Industrial District South Lüttich.) Münch. med. Wochnschr., vol. 78, 1931, p. 49.

13. FENNER, G. Zur Nebelkatastrophe in Industriegebiet südlich von Lütti (Regarding the Fog Catastrophy south of Lüttich.) Chem.-Ztg., vol. 1931, pp. 69-70.

14. ROHOLM, KAJ. The Fog Disaster in the Meuse Valley, 1930: A Fluorine Intoxication. Jour. Indust. Hyg. and Toxicol., vol. 19, 1937, pp. 126-137.

15. MOLLER, P. FLEMMING, and GUDJONSSON, SK. V. Massive Fluorose der Knochen und Bänder (Fluorose bei Kryolitharbeitern). (Massive Fluorosis of the Bones and Ligaments.) Reicharbeitsbl., vol 13 (NF), 1933, p. III 265. (Quoted by Roholm, ref. 3.)

16. SLAGSVOLD, L. Fluoroforgiftning. Norsk Veterinaer-Tidsskr., vol. 46, 1934, p. 2. (Quoted by Roholm,ref. 3.)

17. LILLEENGEN, K. Mikroskopisk undersolgelse av benpreparater fra sauer lidende av kronisk fluorforgiftning. Norsk Veterinaer-Tidsskr., vol. 46, 1934, p. 68. (Quoted by Roholm, ref. 3.)

18. BARDELLI, P., and MENZANI, C. Ricerche sulla fluorosi spontanea dei ruminanti. Nota preventiva. Ann. d'Igen., vol. 45, 1935, p. 399. (Quoted by Roholm, ref. 3.)

19. BLACK, G. V., and McKAY, F. S. Mottled Teeth: An Endemic Developmental Imperfection of the Enamel of the Teeth Heretofore Unknown in the Literature of Dentistry. Dent. Cosmos. vol. 58, 1916, pp. 129, 627, 781, and 894. (Quoted by Roholm, ref. 3.)

20. McCLURE, F. J. A Review of Fluorine and Its Physiological Effects. Physiol. Rev., vol. 13, 1933, pp. 277-297.

21. VELU, H. La Darmous (ou Dermes). Arch. Inst., Pasteur d'Algerie, vol. 10, 1932, 41 pp.

22. KAMINSKI, ARTHUR. Über Schädigungen der Zähne und des Mundes in der Glasindustrie und Berucksichtigen der übrigen gewerblichen Schädigungen. Inaug. Dissertation for Doctor of Dentistry. Breslau. 1928, 26 pp.

23. GREENWOOD, D. A. Fluoride Intoxication. Physiological Review, vol. 20, 1940, pp. 582-616.

24. MARKWITH, R. H. Fluorine and Its Compounds. Ohio Department of Health, 1940, mimeographed pamphlet, 13 pp.

25. EDWARDS, JUNIUS DAVID, FRARY, FRANCIS C., and JEFFRIES, ZAY. The Aluminum Industry. Aluminum and Its Production. 1930, 358 pp.

BUDGEN, N. F. Aluminum and Its Alloys. London. 1933, 278 pp.

26. BRAY, JOHN L. Non-ferrous Production Metallurgy. New York. 1941. pp. 33-34 and 36-38.

27. FEIL, ANDRÉ. Le fluorisme professionel. Intoxication professionnel par l'acide fluorhydrique. (Occupational Fluorism. Occupational Poisoning by Hydrofluoric Acid.) Paris med., vol. 77, 1930, pp. 242-248.

28. GELMAN. (From the Obuch Institute of Occupational Diseases, Moscow.) Jour. Indust. Hyg. and Toxicol., vol. 18, 1936, pp. 371-379.

29. WEBER, H., and ENGELHARDT, W. Über eine Apparature zur Erzeugung niedriger Staubkonzentrationen. Anwendung bei der Untersuchung von Staub aus der Berylliumgewinnung. Zntrlbl. f. Gewerbehyg., vol. 10, 1933, p. 41. (Quoted by Gelman, ref. 28.)

30. ZAMAKHOVSKAYA, E. M. Sanitary-hygienic Estimation of the Composition of the Surrounding Air During the Extraction of Beryllium. Works of the Obuch Institute, Moscow, 1934. (Quoted by Gelman, ref. 28.)

MARTSINSKOVSKY, B. I., and SYROECHKOVSKY, E. E. On the Diagnosis, Clin and Working Prognosis of Severe Poisoning by Beryllium Oxyfluoride. Works of the Obuch Institute, Moscow, 1934. (Quoted by Gelman, ref. 28.)

31. HYSLOP, FRANCES, PALMES, EDWARD D., ALFORD, WILLIAN C., MONACO, A. RAL and FAIRHALL, LAWRENCE T. The Toxicology of Beryllium. U. S. Pub. Health Serv., National Institute of Health Bull. 181, 1943, 56 pp.

32. SHILEN, JOSEPH, GALLOWAY, A. E., and MELLOR, JOSEPH F. Beryllium Oxid From Beryl - Health Hazards Incident to Extraction. Industrial Medicine, vol. 13, 1944, pp. 464-469.

33. WILLIAMS, CHARLES R. Atmospheric Contamination From the Casting of Magnesium. Jour. Indust. Hyg. and Toxicol., vol. 24, 1942, pp. 277-

34. IPATIEFF, V. N., and PINES, H. U. S. Patent No. 2,236,099. (Quoted b Gerhold, et al, ref. 8.)

35. GROSSE, A. V., and LINN, C. B. U. S. Patent No. 2,267,730. (Quoted b Gerhold, et al, ref. 8.)

36. OIL AND GAS JOURNAL. Catalytic Refining of Aviation Fuels. Vol. 43, 1944, p. 81.

37. VAN WINKLE, MATTHEW. Aviation Gasoline Manufacture. 1944, 275 pp. T Hydrofluoric Acid Alkylation Process, pp. 168-173.

38. BENSON, ROY. Hydrogen Fluoride Exposure - Prevention, in the Operati of HF Alkylation Plants. Indust. Med., vol. 13, 1944, pp. 113-117.

39. DRINKER, PHILIP, and NELSON, KENNETH W. Welding Fumes in Steel Fabrication. Indust. Med., vol. 13, 1944, pp. 673-675.

40. READY, N. E. Fluoride Vapors and Dust. California Safety News, vol. 1943, p. 13.

41. WOLFE, WILLIAM A., and KERR, ELIZABETH G. The Composition of Human B in Chronic Fluoride Poisoning. Am. Jour. Med. Sci., vol. 195, 193 pp. 493-497.

42. ROCKWOOD, NATHAN C. Cement Plant Making "Bone Meal." Rock Products, vol. 47, 1944, pp. 44-47.

43. OIL, PAINT, AND DRUG REPORTER. Phosphate Rock Feed. Vol. 146, 1944, p. 7.

44. __________. Fluorine Spray Residue Limit to be Set. Same ref. as above, p. 36.

45. McNALLY, WILLIAM D. Toxicology. 1937, pp. 91-108.

46. SOLLMANN, TORALD. A Manual of Pharmacology. 1940, pp. 853-855.

47. MOLLER, P. FLEMING. Fluorine Poisoning, Seen From the Röntgenological Standpoint. Brit. Jour. Rad., vol. 12, 1939, pp. 13-20.

48. AST, DAVID BERNARD. The Caries-Fluorine Hypothesis and a Suggested Study To Test Its Application. Public Health Reports, vol. 58, 1943, pp. 857-879.

49. McKAY, F. S. Mottled Enamel: The Prevention of Its Further Production Through a Change of Water Supply at Oakley, Idaho. J. Am. Dent. Assoc., vol. 20, 1933, p. 1137. (Quoted by Ast, ref. 48.)

50. CHURCHILL, H. V. Occurrence of Fluorides in Some Waters of the United States. J. Ind. and Eng. Chem., vol. 23, 1931, p. 996. (Quoted by Ast, ref. 48.)

51. SMITH, M. C., LANTZ, E. M., and SMITH, H. V. The Cause of Mottled Enamel, A Defect of Human Teeth. Univ. of Arizona College of Agric., Agric. Exp. Sta., Tech. Bull. no. 32, 1931. (Quoted by Ast, ref. 48.)

52. BALOZET, L., and VELU, H. Bull. Soc. Path. Exot., vol. 24, 1931, p. 848. (Quoted by Ast, ref. 48.)

53. BRANDL, J., and TAPPEINER, H. Ueber der Ablagerung von Fluorverbindungen im Organismus nach Fütterung mit Fluornatrium. (Regarding the Deposition in the Organism of Fluorine Compounds after Feeding with Sodium Fluoride.) Z. Biol., vol. 28, 1891, p. 518. (Quoted by Roholm, ref. 3.)

54. MACHLE, WILLARD, and KITZMILLER, KARL. The Effects of the Inhalation of Hydrogen Fluoride. II. The Response Folowing Exposure to Low Concentrations. Jour. Indust. Hygiene, vol. 17, 1935, pp. 223-240.

55. MACHLE, WILLARD, and EVANS, E. E. Exposure to Fluorine in Industry. Jour. Indust. Hyg. and Toxicol., vol. 22, 1940, pp. 213-217.

56. GAUTIER, A. Sur le role et etat du fluor dans l'economie animale. (On the Role and Condition of Fluorine in the Animal Economy.) Compt. rend. acad. sci., vol. 158, 1914, p. 159.

Original from THE OHIO STATE UNIVERSITY

GAUTIER, A. Sur le role du fluor dans les tissus animaux. (On the Role
Fluorine in Animal Tissues. Compt. rend. soc. biol., vol. 76, 1914,
p. 107. (Quoted by Greenwood, ref. 23.)

57. SHARPLESS, G. R., and McCOLLUM, E. V. (Quoted by Greenwood, ref. 23.)

58. McCLURE, F. J. Fluorides in Food and Drinking Water. U. S. Public
Health Service, National Institute of Health Bull. 172, 1939, 55 pp.

59. __________ Fluoride Domestic Waters and Systemic Effects. 1. Relation
to Bone Fracture Experience, Height, and Weight of High School Boys
and Young Selectees of the Armed Forces of the United States. Pub.
Health Rep., vol. 59, 1944, pp. 1543-1558.

60. ARMSTRONG, W. D. Fluorine Content of Enamel and Dentin of Sound and
Carious Teeth. J. Biol. Chem., vol. 119, 1937, V-VI Proc.

__________ and BREKHUS, P. J. Chemical Composition of Enamel and Denti
J. Dental Research, vol. 17, 1938, p. 27. (Quoted by McClure, ref.

61. DEAN, H. T. Endemic Fluorosis and Its Relation to Dental Caries. U. S
Public Health Reports, vol. 53, 1938, p. 1443. (Quoted by Ast, ref.

62. KNUTSON, JOHN W., and ARMSTRONG, WALLACE D. The Effect of Topically
Applied Sodium Fluoride on Dental Caries Experience. U. S. Public
Health Reports, vol. 58, 1943, pp. 1701-1715.

63. INDUSTRIAL MEDICINE. Caries-Fluorine Demonstration. Vol. 13, 1944,
pp. 561 and 568.

64. AMERICAN JOURNAL OF PUBLIC HEALTH. Michigan Begins Fluorine-Dental
Caries Program. Vol. 34, 1944, p. 1126.

65. DEATHRIDGE, C. F. Fluorine Domestic Waters and Dental Caries Experien
in 2026 White Illinois Selective Service Men. Jour. Dent. Res., vol
22, 1943, pp. 129-137. From abstract by Robert Weaver in Bull. of
Hyg., vol. 18, 1943, pp. 919-920.

66. LINSMAN, J. F., and McMURRAY, C. A. Fluoride Osteosclerosis From Drin
ing Water. Radiology, vol. 40, 1943, pp. 474-483.

67. SMITH, MARGARET CAMMACK, and LEVERTON, RUTH M. Comparative Toxicity o
Fluorine Compounds. Indust. Eng. Chem., vol. 26, 1934, pp. 791-797.

68. DEAN, H. T., SEBRELL, W. H., BREAUX, R. P., and ELVOLVE, E. Effect of
Various Amounts of Sodium Fluoride on the Teeth of White Rats. U. S
Public Health Rep., vol. 49, 1934, p. 1075.

69. BOWES, J. H., and MURRAY, M. M. A Chemical Study of Mottled Teeth Fro
Malden, Essex. Brit. Dent. Jour., vol. 60, 1936, p. 556. (Quoted by
McClure, ref. 58.)

70. LARGENT, E. J., MACHLE, WILLARD, and FERNEAU, IVAN F. Fluoride Ingestion and Bone Changes in Experimental Animals. Jour. Indust. Hyg. and Toxicol., vol. 25, 1943, pp. 396-408.

71. MACHLE, WILLARD, SCOTT, E. W., and LARGENT, E. J. The Absorption and Excretion of Fluorides. Part I. The Normal Fluoride Balance. Jour. Indust. Hyg. and Toxicol., vol. 24, 1942, pp. 199-204.

72. MACHLE, WILLARD, SCOTT, E. W., and LARGENT, E. J. The Metabolism of Fluoride. Ind. Med., vol. 11, 1942, pp. 288-289.

73. JACOBS, MORRIS B. The Analytical Chemistry of Industrial Poisons, Hazards and Solvents. Interscience Publishers, Inc., New York, 1941, pp. 310-315.

74. WILLARD, H. H., and WINTER, O. B. Volumetric Method for Determination of Fluorine. Ind. Eng. Chem., (Analytical Ed.), vol. 5, 1933, p. 7.

75. OCCUPATIONAL HAZARDS. Harrold Fluoride Tester. Vol. 6, 1944, p. 82.

76. NATIONAL SAFETY NEWS. "Iron Lungs" Inhale the Dust. Vol. 50, 1944, p. 92.

77. GERHOLD, C. G., IVERSON, J. O., NEBECK, H. J., and NEWMAN, R. J. The Development of the Hydrogen Fluoride Alkylation Process. Nat. Pet. News, vol. 36, 1944, pp. R-146-R-155.

78. MANUFACTURING CHEMISTS' ASSOCIATION OF THE UNITED STATES. Unloading Anhydrous Hydrofluoric Acid from Tank Cars (ICC Spec. 105 A Series). Recommended Practice. Sheet H-2, 1944, 6 pp.

79. SCHRENK, H. H. List of Respiratory Protective Devices Approved by the Bureau of Mines. Bureau of Mines Inf. Circ. 7237, 1943, 14 pp.

80. SCHRENK, H. H., and PEARCE, S. J. Selection, Use and Maintenance of Respiratory Protective Devices. Bureau of Mines Inf. Circ. 7236, 1943, 12 pp.

81. JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION. Pulmonary Edema Due to Gas Poisoning. Vol. 125, 1944, p. 313.